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INTERNATIONAL CLINICS

A QUARTERLY

OF

ILLUSTRATED CLINICAL LECTURES AND
ESPECIALLY PREPARED ARTICLES

ON

MEDICINE, NEUROLOGY, SURGERY, THERAPEUTICS, OBSTETRICS, PÆDIATRICS, PATHOLOGY, DERMATOLOGY, DISEASES OF THE EYE, EAR, NOSE, AND THROAT, AND OTHER TOPICS OF INTEREST TO STUDENTS AND PRACTITIONERS

BY LEADING MEMBERS OF THE MEDICAL PROFESSION
THROUGHOUT THE WORLD

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Therapeutics

TREATMENT OF TYPHOID FEVER.

A LECTURE IN THE COURSE ON THERAPEUTICS, DELIVERED AT YALE MEDICAL SCHOOL, NEW HAVEN, CONNECTICUT.

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GENTLEMEN,—It is especially appropriate to speak of typhoid fever at the present time, as it is during the autumn months, particularly October and November, that the disease is most frequent. It is often noted as occurring in individuals who have returned only a short time before from vacations in the country. But, while the fall of the year seems to furnish the greatest number of cases of typhoid fever, it may occur at any time, and every large city has constantly some cases of it.

We must first of all diagnose the case and report it to the Board of Health, and assist them, when possible, to ascertain the cause of infection. This is generally in the milk or water supply. As a rule, however, it will be found in city life that the patient was not infected at home, but that the infectious milk or water was taken in some other part of the city, or perhaps in an entirely different part of the country.

Typhoid fever is preventable, and if the excreta of every case be properly cared for the disease will eventually be eradicated. For a number of years now the fæces of typhoid patients have been rather carefully disinfected by most physicians; at least, instructions to this effect have always been given. We have learned, however, that for weeks after the establishment of convalescence the urine may still contain typhoid bacilli. The bacilli from fæces or urine will eventually grow if they are deposited in proper media. They are not killed by freezing or by hot water; only boiling the

germs for fifteen or twenty minutes assuredly deprives them of life. In view of the abundance of this infectious matter, it is easy to understand the necessity for warning the family of danger as soon as typhoid fever is even suspected. The first precaution that must be insisted on is absolute cleanliness on the part of those in attendance on the sick person. The matter of prophylaxis, to prevent the spreading of infection from the case under treatment, is one of the most important elements in the general subject of the treatment of typhoid fever. The patient should not rise, or even sit up, for movements of the bowels or for urination. The stools and urine should be received into a bedpan and urinal, which should contain a solution of corrosive sublimate of the strength of at least one to five hundred. This sublimate solution should be made from tablets that contain certain adjuvant salts which prevent the formation of an albuminate of mercury. If corrosive sublimate alone be used, all albuminous material becomes covered with a layer of this organic salt of mercury, which prevents the corrosive action of the bichloride. The excreta must stand for at least half an hour in the solution; if there are large fecal masses, they must be broken up by stirring, since the mercuric chloride is not able to penetrate to the interior of such masses except after a very long period, and so cannot exert its antiseptic action.

To be absolutely sure, even after this time, that the germs are killed, the fæces and corrosive sublimate should be covered with unslaked lime, or with chlorinated lime. The excreta may then be poured into a water-closet, or if in the country they may be buried with unslaked lime in a place somewhat distant from the house; the location chosen should not be in the neighborhood of a water-course of any kind, or in the general direction from which the wells or springs of the vicinity draw their water. The bedpan or other receptacle should then be cleaned with boiling water, and some of the antiseptic fluid should be poured into it, to make it fit for further use.

All clothing that comes in contact with the patient should be boiled for at least twenty minutes before it can be considered free from germs. To secure absolute safety, such clothing should stand for an hour or more in a weak solution (two per cent.) of carbolic acid. Blankets and woollen underclothing may be subjected to any treatment that seems best; as they cannot be boiled, the carbolic

solution, followed by ordinary washing, will be sufficient. The use of intense dry heat constitutes the most efficient sterilizing method. After complete convalescence, the room may be fumigated, though there is no such necessity for fumigation as after diphtheria or scarlet fever.

An untrained nurse should, of course, be given directions as to absolute cleanliness and the most scrupulous care in thoroughly washing her own hands before she eats. There is probably no infection carried by the patient's breath, but the nurse should be very careful not to inhale the odors or vapors from the excreta. If dry germs are in the atmosphere of the room they may be inhaled, and once in the mouth they may be swallowed and find their way into the digestive tract.

The treatment of the patient in typhoid fever may be considered under the following heads: the general care, the diet, the management of the temperature, the regulation of the bowels, the necessary stimulation, and the treatment of annoying symptoms or complications.

General Care of the Patient.—The first requisite is a large, well-ventilated room, heated by a fireplace if possible. The bed must not be too large, nor should the springs be too lax, for it must be remembered that muscle-aches and backaches are due to sinking down in the bed; the tendency to bed-sores also is less where the bed is level and the pressure more equally divided. The mattress should be perfectly smooth, of course, and present no ridges. The bed covering should be light; if the patient is cold or collapsed, blankets should be used rather than heavy-weight comfortables or spreads or other fancy bed covers. The nurse should have a cot or couch in a remote corner if the room is large, or in an adjacent room if the apartment is small. Two care-takers are almost absolutely necessary for a typhoid fever patient, as the attendance must be constant, night and day. There is no knowing at what moment the patient may decide to get up and walk.

The regular cleansing of the body constitutes a part of the nursing, and in typhoid fever this is generally effected by the spongings ordered. The mouth requires special attention to prevent sores from accumulating on the lips, gums, and tongue. The careful brushing of the teeth and washing of the mouth with an antiseptic wash, containing among other ingredients boric acid,

or even formalin, will generally prevent fissures and erosions. If erosions occur, swabbing the spots with peroxide of hydrogen solution is the best treatment. If fissures or ulcerations occur, touching the parts every other day with a twenty-five per cent. solution of nitrate of silver will stimulate them to heal; if the spot be small, one or two applications of the nitrate stick will be effective.

Diet.—It must be remembered that typhoid fever tends to recovery; but the most important factor in that tendency is the maintenance of the strength. In this protracted fever, in which the waste constantly exceeds the nutrition, the good condition of the stomach is a paramount factor; if the stomach rejects or refuses nutriment, the fight may be harder or the case may be lost, so that our first thought should be never to put any drug into the stomach which will cause irritation. As to food, although there has been a great deal of discussion, the preponderance of opinion seems to be that cow's milk, plain or modified, is the best. Nutriment should rarely be given oftener than every three hours, but this should continue day and night unless a restful sleep or some condition of the stomach postpones it. If the nourishment taken is very little, not more than an ounce or so, as in the case of a child, the frequency should be increased to every two hours. The milk may be given hot or cold. It is best sipped slowly, and must not be swallowed rapidly, to prevent the forming of large curds in the stomach; a tablespoonful of lime water to each glass of milk will also act as a preventive. If milk, as such, is rather disagreeable to the patient, it may be made to sparkle by mixture with one-quarter or one-third part of Vichy, which also furnishes a grateful alkali. Sometimes a pinch of salt to a glass of hot milk changes its character, and the salt is always of advantage. Koumiss may be substituted if the patient likes a sour taste to the milk; it is generally well borne by the stomach and is nutritious. Where rich milk disagrees, skimmed milk may be given, which has also the advantage that the patient is thus drinking large amounts of water for flushing purposes. Of course, for sufficient nutrition on a skimmed-milk diet, considerable bulk must be given, at least three quarts in twenty-four hours.

As additions to the diet, or substitutes, malted milk, various prepared albuminous foods, beef peptones, and beef teas may be used; or we may give the white of an egg, well beaten with an equal part of water, with perhaps the addition of a little brandy. Ordi-

nary beef tea will furnish stimulation, not nutrition, and may be added to the diet. We may press the juice from beef and administer it sometimes, remembering that we get not only the blood extracts, but also muscle juice by this method, which has lately, under the name of zomotherapy, proved to be a promising treatment for tuberculosis.

The amount of nutriment should be carefully recorded on the symptom records, and you should judge whether or not your patient is receiving sufficient. An adult should take not less than three pints nor more than two quarts of milk, or its equivalent, in the twenty-four hours. Any gastric distress or much gastric flatulence should cause you to rearrange, change, or modify the diet. Let me caution you against overfeeding in typhoid fever. This you are apt to do in the first week, at which time, while the fever is sthenic and rising, you can make the amount of nutriment a little less than later. Be sure also that the patient is thoroughly aroused before the nutriment is given, else there is danger of some liquid getting down the trachea and causing pneumonia.

All fever patients should drink a large amount of liquid. This tends to improve or increase perspiration, to keep the skin moist and in good condition, and to increase the bulk of urine, which is particularly desirable, as the kidneys easily become congested in fever cases and excrete a small amount of very concentrated urine. Plain water or distilled water has been given typhoid patients in large quantity with the belief that this large amount of liquid increases the action of all the eliminative organs and lowers the fever. A typhoid patient may be so apathetic or stuporous as not to ask for water, but liquid should be frequently given whether asked for or not. Aërated waters may be given, and acid drinks, as lemonade, or water acidulated with dilute phosphoric, hydrochloric, or sulphuric acid, are all allowable; perhaps none is better than lemonade. Barley water is the most soothing to the mucous membranes, at the same time carrying with it a small amount of nutrition; it makes, theoretically, the best drink we can furnish. A tablespoonful of prepared barley is cooked in a pint of water, which is then strained and cooled, and furnishes a very bland liquid. Cracked ice may, of course, be eaten *ad libitum,* or tablespoonful doses of ice-water may be given.

Begin to give tincture of the chloride of iron pretty early in

every case of typhoid fever. I have found it in practice of marked benefit in rendering the whole course of the fever less active, with lower temperature and with fewer complications, and in theory anything that will improve the condition of the blood will render the patient better able to fight the disease. The drug seems to aid the normal germicidal action of the gastric juice, which probably is always diminished during intense fevers; it may thus prevent the multiplication and migration of active bacilli from the intestine into the stomach, and you may remember that even the colon bacillus has been found in the stomach. As a germicide for the typhoid bacillus the chloride of iron is exceedingly active, even in very dilute solutions, and I believe it to be of benefit in typhoid fever, whether the principal element of good in its action after absorption lies in its increasing the antitoxic power of the blood serum or in stimulating the hæmatopoietic organs. You may administer it by itself in fifteen-drop doses, well diluted, at six-hour intervals, after the nutriment, or you may combine it in a prescription, as follows:

℞ Tr. ferri chloridi, f℥i;
Syr. acidi citrici, f℥iii.
Sig.—Teaspoonful, well diluted, every six hours.

Or you may add dilute phosphoric acid in equal amount to the tincture of the chloride, and administer as the other preparations.

Management of the Temperature.—A temperature of 105° or even 106° F. for a short period, as that caused by a malarial paroxysm, is not dangerous to life or to the organism; but a long-continued high temperature, even though very much lower, is dangerous by reason of the increased metabolism and the fatty degeneration which it causes. Various dogmatic limits have been named above which we should endeavor to prevent the temperature from rising in typhoid fever. As a working rule, except perhaps for the first day or two, during which you are getting the patient and his symptoms under control, the temperature should not go above 102° F., at least for any length of time; you then decide to use antipyretic measures whenever the temperature reaches 102° F. or over. In the first day or two, especially if there is severe headache, a migraine tablet, composed of acetanilid (generally two grains), monobromated camphor (about half a grain), and caffeine (about half a grain), should be administered, and may be repeated in three

hours if there is no improvement. This remedy may be given in this manner for two or three days; but the treatment should not, as a rule, be kept up long. Some other antipyretics of similar nature may be used; phenacetine is perhaps the best, in five-grain doses, given in tablet or capsule. If the fever begins very high and the stomach is in good condition, you may give a gramme (fifteen grains) dose of antipyrin, in two capsules, each containing half a gramme, followed by a good large drink of water. This, however, produces profuse sweating, and should not be repeated.

The very best agent for reducing the temperature in typhoid fever is, as you well know, cold water in some form. Currie, of Liverpool, in 1797, and Nathan Smith, who was professor of practice here at Yale from 1813 to 1828, at about the same time introduced the cold-water treatment of typhoid fever. This was forgotten until reintroduced by Brand in 1861, and was then again forgotten; it was finally re-established about five or six years ago under the title of the Brand treatment of typhoid fever. With the details of this treatment you are already familiar. The intensity or the pertinacity of the fever decides which method of cold application shall be made. The mildest is, of course, sponging with tepid water. From this we progress through sponging with cold alcohol, sponging with cold water, sponging with ice-water, use of ice-water bags, cold-water and ice-water coils, especially to the abdomen and to the head, cold compresses, wet sheets, cold sprinkling, and finally the cold bath, or tubbing.

The real Brand method consists in immersing the patient in a bath at a temperature of 85° F., or even as low as 75°, allowing him to remain until his temperature falls to 100.5° or 101° F., and repeating every three hours as long as the temperature is above 102°. Tubbing requires at least three assistants to do it well, and usually your personal attendance to see that the temperature is not lowered too far and to note whether shock is caused. The thermometer should be kept constantly in the rectum during tubbing, and must be read every minute or two. This treatment should be used, especially in private practice, only when all other methods have failed; I believe you will find it rarely necessary. Positive contraindications to tubbing in typhoid fever are, in my opinion, such complications as pneumonias, intestinal hemorrhage, menstruation, nephritis, and cardiac complications. If I may be par-

doned the Irish bull, I wish to emphasize the fact that the best treatment of high temperature in typhoid fever is to prevent it.

A large amount of this fever is due to secondary infection. By this I mean that there is, besides the fever caused by the typhoid bacillus and its toxins, that caused by all kinds of absorbed toxins from intestinal putrefaction, decomposition, and fermentation. We must not forget that in typhoid fever there are ulcerations in the lower part of the small intestine. These are like any other surgical ulcerations, and the cleaner we can keep the parts involved the lower will be the temperature. The better washed out the bowels are, the less will be the absorption of other products from these ulcerations; and the better the condition of the bowels, the better will be the action of the liver, the less the congestion to the portal circulation, and again the less absorption of toxins, not only from other sources, but from the typhoid bacilli also. We can readily see that with the pressure always negative in the intestines there would be less tendency to absorption and migration of typhoid bacilli; and we know by the complications that these can migrate into almost any part of the body, having been found everywhere. Disturbances here can also cause the colon bacillus to change its habitat, resulting in the general disturbance of the system. Hence, bowel cleanliness, as near as we can assure it, is the basic treatment in typhoid fever.

Management of the Bowels.—We thus come to the fourth indication, or the management of the bowels. Make it a working rule that in this disease, which has diarrhœa as a symptom, caused by inflammation and irritation in the intestine, constipation is something undesirable and to be prevented. Bear in mind also that a troublesome, fretty diarrhœa with small movements is not what is desired, but a daily complete and thorough evacuation, unless there has been considerable hemorrhage. I do not believe that there is any specific to cause these evacuations. The much-lauded Woodridge treatment of small doses of combined cathartics frequently repeated has its advocates, and tends to the result we desire. The calomel treatment has also its adherents, and I believe it does good, especially in the beginning of typhoid fever. Give it in sufficient doses, perhaps thirty centigrammes (five grains) combined with bicarbonate of sodium one gramme (fifteen grains), every other day during the first week, if it does not cause depression.

Castor oil in capsules, or in combination with saccharin and oil of wintergreen, is also a good laxative; this, however, is liable to disturb the stomach. Later on, after the end of the first week, calomel being too active a cathartic, in my opinion at least, I believe that a gentle saline is the best treatment. A dose of Carlsbad salts, Hunyádi water, or, if necessary, Rochelle or Epsom salts in amounts to suit the patient, may be given daily. If the stomach is disturbed, or tends to refuse these laxatives, glycerin and water enemata, a tablespoonful of glycerin to four tablespoonfuls of water, will almost invariably give satisfactory results. Leave some one-tenth grain morphine tablets with the nurse, with directions to administer one of them each day after there has been one large movement or two small ones, to stop any persistent intestinal evacuation.

Now as to intestinal antiseptics. The Philadelphia treatment, carbolic acid one drop and tincture of iodine two drops to a glass of water every few hours, I believe not to be advisable, because of the tendency of such remedies to disturb the stomach. None of these liquid antiseptics will ever reach the lower part of the small intestine, and they are too weak to be of use to the stomach. There are several bowel antiseptics, of which I believe salol to be the best, given in doses of twenty centigrammes (three grains), for the first few days at three-hour intervals, and after a week at six-hour intervals. This dose is small, but if the dose is too large the continuance of the treatment will be followed by kidney irritation. During the administration of salol the nurse should always note the color of the urine, reporting if it becomes dark, showing too much carbolic acid, and you should occasionally also examine it for albumin; either dark or albuminous urine should be the signal to stop the salol. Beta-naphtol bismuth is also given as an antiseptic, as are beta-naphtol itself and thymol, but I believe salol is the best.

With this treatment to prevent intestinal fermentation, and with the daily evacuation of the bowels removing débris and particles of decomposition, you will find that you have less fever from secondary infection, and that you have done away with the troublesome and dangerous tympanites, which so many times cause erosions enough for hemorrhage, or even perforation and its resulting peritonitis. The typhoid symptoms also are less,—that is, the nervous symptoms, the delirium, stupor, and mental hebetude. To

take a case of typhoid fever through its course without hemorrhage, without delirium, and without troublesome tympanites, is a credit to any treatment. Of course, any great weakness of the pulse, or hemorrhage from the intestine, or symptoms of peritonitis, dictates the omission of the daily dose of laxative.

Stimulation.—Alcohol has been used from time immemorial for stimulation in typhoid fever; there are, however, many advocates for the non-alcoholic treatment. Let us see what we believe to be the object of alcohol. In the first place, it is a food in the sense that it furnishes something for the patient to burn; this high temperature causes all metabolism to be quickened, and he must burn something. He burns first his fat, then his muscles, and then we begin to have not only wasting, but actual degeneration. Alcohol, then, will prevent some of this loss.

But for my part, I believe the reason alcohol has received such commendation in this disease is because of its secondary effect, in dilating the peripheral blood-vessels. When the administration of alcohol is pushed this always occurs, tending to increase the perspiration, with a resulting increase of evaporation and radiation, and a consequent loss of heat and lowering of temperature. We also note a distinct tolerance for alcohol in this disease, perhaps because so much of it is quickly burned up. We find that a very rapid heart is relieved and is made slower by alcohol, and that in normal conditions a slow heart is made rapid. If the pressure is too great, alcohol will dilate the blood-vessels in ordinary conditions and relieve the tension. Alcohol certainly tends, then, to reestablish the equilibrium of the circulation. The rapid pulse of a typhoid case will become slow if the proper dose of alcohol is given.

The indications for alcohol in typhoid fever are rapid heart, high fever, and typhoid head symptoms, *i.e.,* low muttering delirium, subsultus, carphology, trembling tongue, and dry skin. A pulse of 110 in an adult is on the verge of becoming weak, and one of 120 is a positive indication for stimulation in typhoid fever; alcohol is indicated also by a pulse below 100 that is of low tension and perhaps dicrotic. The size and frequency of the dose depends entirely upon the results, coupled with the past history of the patient; an alcoholic patient or one used to considerable alcohol requires, of course, more than others. Brandy and whiskey are the forms in which it is best administered, beginning with teaspoonful

doses every three hours; it may be given diluted with water, milk, effervescing water, or albumin water, as seems best to suit the case. Beware of giving too much alcohol; I believe that in very many cases of typhoid fever the meningeal symptoms are practically delirium from alcohol. If, while you are giving alcohol and pushing it, the pulse becomes strong and bounding, the skin and tongue dry, the face flushed, and the breath gives off the odor of alcohol, you are overdosing the patient. The character of the pulse, then, decides when you should begin the use of alcohol; but I believe that in any ordinary case the patient is benefited by a little alcohol. Remember that, while you may give alcohol in conditions of collapse, it is not to be relied on as the treatment, as you can easily cause too much vasomotor dilatation with large doses of it.

I find that in certain cases I can replace alcohol with small doses of nitroglycerin. In the first part of typhoid fever, perhaps the first two or two and one-half weeks, small doses, as one two-hundredths of a grain every six hours, have seemed to me to have very good results in lowering the temperature, relieving the pressure in the head, and obviating the necessity for much alcohol. Sometimes I give this in the Da Costa tablet, which is a combination of two minims of tincture of digitalis, two minims of tincture of strophanthus, and one-quarter minim of tincture of belladonna, with one two-hundredths of a grain of nitroglycerin. If the pulse is weak, this makes an excellent combination.

At any time during the course of typhoid fever, though we do not expect it until the latter part of the third week, we may begin to find a weakened heart and general depression. The decrease of the temperature is the signal for the administration of strychnine, and also, perhaps, for some increase in the alcohol; it is best to reserve the giving of strychnine until such a condition supervenes. During this period, especially in the early part of the day, doses of black coffee also are stimulating and decidedly good for the heart. The dose of strychnine depends, of course, on the indications. One-thirtieth of a grain tablet (two milligrammes) every six or eight hours is a good full dosage; every three hours is too frequent, and if any such quantity of strychnine is necessary, it would better be given hypodermically in one-fortieth grain doses.

Treatment of Troublesome Symptoms or Complications.—This is an important part of the therapeutics of typhoid fever. The

first thing we have to contend with generally is headache, which may usually be relieved by a migraine tablet or two, given the first two or three days. If the fever is high, cold applications to the head will relieve it. Bay rum or cologne-water is very grateful to the heads and foreheads of some patients.

Constipation, as I have said, should be prevented. Diarrhœa can be stopped by gentle catharsis and an occasional one-tenth grain tablet of morphine. A few compressed tablets of bismuth subnitrate or subcarbonate may be given, and sometimes seem to soothe intestinal inflammation. Sleeplessness, or insomnia, in typhoid fever is best combated by the smallest dose of morphine which you find on trial will produce sleep. If there is meningitis, or any meningeal symptoms at all, morphine is contraindicated, and the bromides and cold applications to the head must be used; the bromides must not be given too long, lest they weaken the patient.

If, in spite of your care of the bowels, tympanites supervenes, a cold compress on the abdomen will sometimes cause evacuation of the gas, and glycerin enemata, with possibly a little oil of turpentine, will aid its expulsion. The rectal tube is rarely of value, but should be tried. I wish to reiterate, however, that prevention is the best treatment for tympanites in typhoid fever. If it occurs, it is one of the hardest difficulties to surmount, and the danger to the patient lies not only in the possibility of its causing hemorrhage, perforation, and the absorption of intestinal toxins, but in the pressure it exerts upon the heart, causing a great deal of cardiac disturbance and distress. You cannot be too careful, therefore, in keeping the bowels in good condition and thus preventing this troublesome complication.

If intestinal hemorrhage occurs, as it sometimes does early in cases of walking typhoid, though ordinarily we do not expect it until the third week, the indication is for absolute rest. If the hemorrhage is slight, we stop the food for a few hours, give morphine either by the mouth or hypodermically so as to secure peristaltic rest, and prescribe one of the tannic acid preparations in capsules for its local astringent action. Tannic acid itself being too irritant, we may use protan, a nucleo-proteid with tannin, in half-gramme capsules every three hours, tannigen in the same doses, or tanalbin. Gelatin is used as a coagulant, either given by the mouth in the form of wine jelly, or injected into the subcutaneous

tissue or into the muscles in aseptic solution. I do not believe that ergot is of any value whatever in this condition, but do believe that nitroglycerin, by dilating all of the blood-vessels, will relieve the tension in the internal vessels. If the hemorrhage is severe an ice poultice (cracked ice with sawdust or bran) may be applied to the right iliac fossa, or an ice-bag may be used. Elevation of the foot of the bed prevents restlessness from brain anæmia. If collapse is imminent, do transfusion, or hypodermoclysis with normal saline solution. A sudden drop of temperature, pallid face, and lack of good tone of a pulse that was previously all right, should make you suspicious of hemorrhage even though no blood has yet passed, and your treatment should be the same as though you had found it.

Perforation generally gives more of an indication of its onset. There is a sharp pain referred to some portion of the abdomen, facial anxiety, and increasing abdominal tenderness; the patient is not collapsed as he is from hemorrhage. The immediate treatment is morphine administered hypodermically in doses sufficient to stop all peristaltic movements; then stop all food, and consider the advisability of laparotomy. As any symptoms of beginning peritonitis make a laparotomy almost too late, surgical consultation should be advised as soon as you are convinced or even suspicious that perforation has taken place; the joint decision as to the advisability of operation should be followed. If you are right, and perforation has taken place, it is but rarely that a case recovers, and laparotomy gives the only chance.

Collapse may occur from hemorrhage, or from a weak heart at any time, and the heart may bother you from the beginning of the case to the end. We expect to find an irritable heart without serious complications when the temperature begins to fall, the loss of stimulation from the temperature, with a sudden morning drop, leaving the heart without its support. At this period we are giving fair but not overpowering doses of alcohol, and strychnine has been started. Actual collapse occurring, strychnine and atropine should be given hypodermically, and pushed; a saturated solution of camphor in olive oil should also be given hypodermically. Dry heat should be applied to the feet and body, and especially to the heart. Oxygen inhalations may be given if you see fit. Aromatic spirit of ammonia makes an additional cardiac stimulant of great value.

Nitroglycerin is not indicated, and digitalis, if used, only in very small doses. Caffeine in the shape of coffee is a good addition to your treatment. If large doses of morphine have been given for peritoneal pain, be very careful to see that the urine is evacuated at least every six hours; if not, the catheter should be used. In fact, even without peritoneal pain, and without large doses of morphine, the patient's apathetic condition sometimes allows the urine to be retained. Of course, with a nurse and a symptom record you would notice the absence of this function, but do not fail to inquire in cases where you are not blessed with a nurse.

Prevention is the best treatment for bed-sores. The turning of the patient from one position to another, which is perfectly permissible in this disease, changes the points of pressure, and sponging with alcohol the parts on which the sores most frequently occur, gentle massage with lanolin, and, if reddening occurs, the application of peroxide of hydrogen, dry powders, and various forms of ring-cushions, generally prevent actual ulceration. If ulceration does occur, treat it on surgical principles.

Treatment of Convalescence.—This is, perhaps, the period in which you will be fretted most by the patient and by the family as to when the patient can sit up and when he may have something to eat. Never feel safe from the occurrence of a relapse until your patient has been ten days free from fever. Gradually stop the treatment, except perhaps a small amount of alcohol, the necessary dose of strychnine, and such iron doses as seem proper. Up to this time the diet should be liquid. You then may gradually begin to give gruels, a little more substantial broths, milk toast, poached eggs, and in a day or two a chop, a small piece of steak, or a little croquette of scraped beef, and then gradually on to baked potatoes and a slowly enlarged diet, noting any disturbance of the temperature. As to sitting up, I think the patient can sit up, at least in bed, before he can eat solid food; but this, of course, depends upon the condition of the heart. As soon as convalescence is established, gentle massage for the muscles should be instituted.

The most troublesome symptom at this period is constipation, for which glycerin suppositories or glycerin enemata are very suitable, and later you may resort to cascara or compound aloin tablets.

Ordinarily the hair would better be cut, and some tonic, or perhaps an occasional shampoo with resorcin soap, should be insti-

tuted. After this severe fever the nails are brittle, the hair has a tendency to fall out, and the teeth are prone to decay, showing that there is an absence of phosphates and lime salts. For proper nutrition, one of the elixirs of glycero-phosphates should be given.

An occasional sequela of typhoid is thrombosis, most frequently of the femoral vein. Treat this as you would thrombosis from any other cause, by elevation of the leg, morphine for pain, and a flannel bandage to prevent, if possible, excessive œdema; ichthyol with olive oil (perhaps in equal parts) should be applied to lessen the inflammation. Great care should be taken, as too much manipulation of the legs or applying the bandages too tight may loosen emboli into the blood current, which could cause sudden death, or perhaps even pyæmic complications.

If relapses do occur, they generally tend to recovery, and a true relapse of typhoid fever is a diminutive course of the real disease with the same typical ascension and declination of temperature. Many pseudo-relapses take place, which may be caused by any indiscretion in diet, excitement, or by an autointoxication from the intestine.

TREATMENT OF INTESTINAL PERFORATION IN TYPHOID FEVER.

BY NOËL MAUGER, M.D.,

Of Versailles.

The diagnosis and treatment of intestinal perforation accompanying typhoid fever have recently been under lengthy discussion in the medical and surgical societies of Paris. This discussion was precipitated by accounts of several new cases where recovery followed operation. The reports of these cases were taken up, and both physicians and surgeons gave their opinions thereon. The surgeons were practically unanimous in advocating operating; a number of the physicians still preferred medical treatment. The ordinary practitioner, therefore, hardly knows where he stands in the matter, yet he should form a definite opinion, for he may be called upon at any moment to make a decision between the two treatments. It has seemed wise, then, to mention the published statistics, and, after having shown by figures the advantages of surgical intervention, to give the method of operating by which the best results have been achieved.

To begin with, to avoid the occurrence of perforation, certain measures and precautions must be advised. There can be no doubt that the cold-bath treatment, which has been almost universally adopted in typhoid fever, is, owing to its anti-infectious action, one of the best preventives.

In spite of what has been said to the contrary, during convalescence the diet should be exclusively liquid for at least two weeks after the temperature has dropped to normal. There is no question that errors in diet are a frequent cause of perforation in typhoid. Among many may be mentioned two recent cases. In the first death occurred after eating a piece of bread, in the second after a breakfast roll.

Certain enemata, clumsily administered, and purgatives (of which we should be chary in the third week) have caused accidents, owing to the intestinal contractions to which they give rise.

All forms of effort, coughing, sneezing, voluntary movements, and straining at stool, cannot be too closely guarded against. Meteorism and constipation should be carefully avoided; the former, by repeated applications of compresses dipped in hot water; the latter by intestinal lavages properly administered.

The patient should even be carried from bed to bath-tub to prevent the effort and fatigue attendant upon walking.

But in spite of all these precautions and without being able to define a cause, perforation may occur, and the therapeutic problem is presented. The baths must be given up, the patient kept absolutely motionless, ice prescribed inside and out, and opium administered in generous doses. But of what use can such a line of action be against an intestinal perforation through which gases and fecal matter are constantly passing? Consequently, in forty-eight hours, more or less, death supervenes. Recovery has been reported, but in such small proportion (one in twenty, according to Murchison), and by the aid of such specially fortuitous circumstances, that such cases should be regarded as pathological curiosities rather than as deductive facts. These instances are almost always forms of circumscribed peritonitis starting from the appendix or colon; and the abscess either opens to the outside or into another intestinal loop or neighboring viscus. However, hardly more than twenty such cases are known in medical literature. Encysted peritonitis is rare in typhoid fever, peritoneal generalization being the rule. Cases of recovery of the latter form are still more rare.

Leyden was the first, in 1884, to advise surgical intervention for intestinal perforation in typhoid. And in the same year, Mikulicz published a case of perforation cured by operation. Soon after, reports of four other cases followed from different surgeons.

In 1890 Louis collated eleven cases of operative intervention, of which two had recovered.

In 1896 Lejars published two cases of his own. At that date there were in all twenty-five known cases with six recoveries.

In the same year, in a thesis on surgical intervention in acute, diffuse peritonitis, Houzé showed the disorder to be frequently caused by typhoid fever, and cited twenty-eight cases with six recoveries. [By 1897 the figures had reached thirty-one cases with seven recoveries, and shortly after statistics showed sixty-five cases with twelve recoveries.]

William Keen, in his excellent work on the sequelæ and surgical complications of typhoid fever, gave eighty-three cases with sixteen recoveries. And Platt, in 1899, in the *Lancet,* added to this list three personal cases ignored by Keen, and fourteen new cases. A further publication in the *Lancet,* 1899, gave one hundred and five cases, with twenty-two recoveries. Carefully selecting for myself from among the cases that came to my notice (both published and unpublished) only such as were certain, I have been able to collate a list of one hundred and seven, with twenty-five recoveries, or twenty-three per cent.

Final statistics, published in the *Revue de Chirurgie* of last year, gave ninety cases, with sixteen recoveries, a little over twenty-one and a half per cent.

If we compare these latest figures with the results given by medical treatment, we are soon convinced that surgical intervention in typhoid perforation is fully warranted for the following reasons.

First, the lesion of the intestines is usually a simple one, since in eighty-two per cent. of the cases there is only one perforation, which fact encourages operation. In only five of the cases published did later perforation compromise the results obtained.

Again, the perforation is easily found, since in eight cases only of all furnished by statistics were the operators unable to do so. Even then the result was favorable with four.

There is also great assistance in the knowledge of the probable point at which the perforation will be found, occurring as it does usually in the latter portion of the ileum, sometimes in the cæcum, and frequently in the appendix. The surgeon, therefore, has only to examine the right iliac fossa for a very limited area.

Surgical intervention is furthermore favored by the rapid agglutination of the tissues and occlusion of the orifice. In Bouet's case the orifice was closed eleven hours after being sutured, and in two cases only has *post-mortem* revealed the sutures to have given way.

The opponents of laparotomy have nothing to offer but impressions in opposition to the foregoing facts. The bad general condition of the patient would certainly be an argument had it not been proved to the contrary, certain patients having undergone two or three operations during their attack. Three of the cases published are specially striking in this respect. In the first, the

patient recovered after having presented a succession of abscesses of the parotid glands and of the cellular tissue, double otitis, and purulent arthritis of the knee. In the second case, a child of ten was operated upon in the space of a few days, first for perforation and again for occlusion. Recovery followed. In Cushing's case, in two weeks a patient successfully underwent three laparotomies, the first for perforation, the others for occlusion.

The argument advanced, relative to the difficulty of making an early diagnosis, has, however, really some weight, since figures show that the earlier the operation the greater the chance of success. In one list of twelve cases of recovery, ten of the operations were performed during the first twenty-four hours, the other two having been operated upon in the twentieth and sixtieth hours respectively.

The correct deduction, then, would seem to be, not that operating should be condemned, but that the diagnosis should be made at the earliest moment possible. To this fact the societies of medicine and surgery have been fully alive. Let us sum up as briefly as possible the elements of this early diagnosis.

To avoid error we must start with the idea that in perforation of this kind we are dealing with a very peculiar form of peritonitis. The lessened resistance of the patient and the prostration present give the evolution of the peritoneal symptoms a very masked character. As a usual thing, they are merely outlined, and one must be satisfied with this and not expect a complete group of symptoms of a peritoneal attack. Though cases have existed where the entire symptomatology was to be found, they are rare. In typhoid perforation sometimes there will be but one symptom that is absolutely unmistakable.

The symptoms may be classed in the following order of importance: pain, pulse, temperature, fæces, tympanites, disappearance of hepatic dulness, muscular defence, bladder trouble, constipation, vomiting, rigor, and air and water sounds.

Pain is a distinctive sign seldom wanting. It may exist alone and be localized, but it exists. Its intensity varies; in some cases it is so severe as to produce syncope; in others, light and resembling intestinal colic.

When the number of pulsations (which is relatively moderate in typhoid fever) suddenly rises, and at the same time the volume

of the pulse diminishes noticeably, the peritoneum should be watched. Pulse frequency and depression have been noted in nearly all histories that have been carefully taken.

When perforation occurs, the temperature generally either rises or falls, more frequently the former; but, as sometimes there is no change at all, this sign is by no means infallible.

The facies is of considerable value to the physician who has watched the case from day to day. If a marked change in the appearance of the patient is suddenly noticed, and this is accompanied by even slight abdominal pain and even moderate acceleration of the pulse, the diagnosis is possible.

Abdominal tympanites is a good sign, is rarely lacking, and can be easily detected.

The issue of intestinal matter and gas may push up the liver and lessen its ordinary dulness. This occurrence is mentioned in several of the histories given, I myself finding it in one case. It should therefore be looked for and credited with the importance due.

Muscular defence is an early symptom, rarely lacking and always reliable. It is first confined to the region of the perforation, but soon extends to all the abdominal muscles.

Bladder disturbance is common, micturition being difficult and painful. Dr. Terrier lays great stress upon this point, as well as upon the cessation of the stools. Both of these disturbances can be readily noted in a typhoid patient.

Rigors may be lacking, and vomiting is a tardy symptom, more than usually tardy in this variety of peritonitis, as its appearance indicates a reaction that is already quite intense.

Examination of the blood will sometimes assist in the diagnosis. The fibrinous reticulum, which in typhoid fever is little altered, will be slow in forming and thickened in intercurrent inflammation. The piles of red globules will form little islands, and the inflammatory patches described by Hayem will appear. The leucocytosis, which, in typhoid fever, is considerably below the normal, will rise when inflammation sets in, and the number of white globules soon goes from five or six thousand to ten.

Lastly, after any suppurating process, the average of the polynuclear globules, usually low in typhoid fever, increases noticeably. William Keen and Finney think that a blood examination might,

in some cases, confirm an uncertain diagnosis of typhoid peritonitis.

A final objection raised by the adversaries of laparotomy, is, that to be forced to make an early diagnosis may be against the patient's interests in that it may lead to unnecessary operation. Let us consider, then, what may be the causes of error.

If the error be nothing more than a mistake as to the nature of the peritonitis, the harm done will be slight, since the surgical treatment is the only one applicable. Variations in temperature, more than anything else, are likely to give rise to mistake because, in typhoid fever, a high temperature occurs with a relapse, which is also accompanied by the usual symptoms of the infection (diarrhœa, headache, and rose spots). It also occurs in appendicitis following typhoid, during convalescence, when McBurney's point will be found. And lastly, it comes into any suppurative complication, which a thorough examination should always be able to detect.

It should also be borne in mind that a very low temperature is found in intestinal hemorrhage (though then it is followed by a rise in temperature with a rather slow pulse); in cases that end with a sudden drop of the fever instead of with lysis as is usual; in certain intercurrent hemorrhages (epistaxis, pulmonary infarctus, menstruation, and miscarriage); in hepatic degeneration under the influence of certain drugs (digitalis and quinine); or in pulmonary complications, as pneumonia or congestion.

The state of the pulse may also lead to error, the prostrated condition of the patient, a small and weak pulse, and faintness of heart sounds led me in one case to mistake perforation for myocarditis.

And, lastly, too much importance should not be attached to excessive tympanites.

I am convinced, however, that operation through error, though always to be regretted, has never done any real harm. There must be at least ten cases of laparotomy performed through mistake without the loss of a patient. It is fair, then, to conclude that when, after an exhaustive examination, the diagnosis is still uncertain and the patient's life is in danger, one is authorized to intervene.

From eight to twelve hours at least should elapse between the time when the perforation occurs and that of the operation. During

this period the patient should be stimulated with injections of ether or caffeine, and particularly with abundant intravenous or subcutaneous injections of serum.

Success depends upon the shortness of the operation, a half hour being the average time. If possible, this should not be exceeded. Knowing the approximate seat of the lesion aids materially in shortening the process.

Either ether or chloroform should be carefully and moderately given, a few drops of the latter usually being sufficient. The spinal injection of cocaine might be found useful.

The incision should be lateral or on the middle line. Both have their advantages, the lateral incision bringing the operation nearer the diseased region, that in the middle allowing a more complete and ready exploration of the whole intestine. (The perforation may not happen to be in the ileum.) Both middle and lateral incisions may be made at once. Large counter-openings facilitate drainage, and some surgeons even advise these large and multiple openings.

Perforation should be methodically looked for. After making sure that the cæcum and the appendix are healthy, the final half-yard of the ileum should be closely scrutinized. It is usually here that the perforation will be readily detected, on account of its wide orifice and the fecal matter escaping from it. The operator should also remember that certain intestinal diverticula may be the seat of perforation.

If the perforation found concerns the appendix or a diverticulum, that organ has simply to be removed, but if the intestine is perforated, the orifice should be sutured. To fasten the perforation to the abdominal wall appears to be only applicable to a few special cases. To resect the intestine is a long operation with serious shock, and the results are no better. Therefore, it should only be performed where there is extensive loss of substance and when neighboring ulcerations threaten to perforate in a short time.

Suturing is therefore the best method, and should be preceded or not, according to its condition, by the removal of the diseased portion. As a general thing, the diseased tissue should be thrust back into the intestine and the serous membrane be sutured by separate stitches, as continued stitches might destroy the intestinal walls, on account of their friability. At least two rows of stitches

should be put in, and three would be better since intestinal stricture is not to be feared.

The neighboring intestinal folds should then be examined and stitches placed in any ulcerations that are threatening to perforate. This should all be done as quickly as is consistent with thoroughness to avoid a prolonged use of anæsthetics.

The cavity should then be washed out with boiled water or with serum at a temperature of about 50° C., in preference to being cleaned with tampons. There is generalized peritonitis, and consequently no fear of any spread from a localized focus. The washing should be very copious. Tampons would naturally be used if there were only circumscribed peritonitis.

Drainage is of the utmost importance and should be carried out by strips of gauze, or, better still, by large tubes. Multiple incision alone provides thorough drainage. Only the ends of the incisions in the abdominal wall should be closed by a few stitches, as the best results have been noted when the wound is left wide open. Then cover with moist, sterilized gauze.

Finally, the patient should be closely watched as to pulse, temperature, and facies. Champagne and serum should be administered, two litres of the latter per day. Tympanites should be remedied as far as possible, and careful watch be kept for intestinal occlusion, which has been reported in some cases.

THE TREATMENT OF MORPHINISM.

LECTURE DELIVERED AT THE NEW YORK SCHOOL OF CLINICAL MEDICINE, NEW YORK CITY.

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GENTLEMEN,—The first object in the treatment of morphinism must be to secure the control of the patient. His volition must be made subservient to that of the physician, and he cannot be allowed to reason or direct as to the plan of treatment. Failure always follows self-treatment. Removal from home is most essential to secure this control; as in other neuroses, particularly insanity, hysteria, and forms of neurasthenia, control by strangers and contact with them are most effectual, helping to break up the morbid trend of reasoning and associations in a manner not possible at home and with relatives. Private and special asylums, if properly managed, have superior advantages which cannot be obtained elsewhere. In such places the stimulating firmness of a stranger, if used with tact, does much to arouse a weakened will. All the influences, exerted, as they are, with the central purpose of discontinuing the morphine, will encourage personal effort on the part of the patient. This idea should dominate all others at the beginning, and no conditions which antagonize this design should be allowed to prevail.

The tendency of each patient is to exaggerate the importance of conditions and surroundings in the treatment, and to consider the process of withdrawal of the drug and final cure as dependent on some insignificant circumstance. This idea is to be antagonized and overcome by the efforts of the physician and attendant. If the patient's mind can be concentrated so as to have full faith in the efficacy of the means used, it is additional help. This follows after a few days' treatment in most cases, and is often the basis of success. If the mind is unsteady and unable to retain confidence in the meas-

ures employed, the physician must be resourceful enough to supply this deficiency.

In some cases the morphinists are continuously casting about for some new means and measures superior to those used; no plan of treatment, however enthusiastically begun, is ever continued long. If the patient is a physician, the difficulty is increased and the treatment is more uncertain. If he can be persuaded to trust implicitly to the physician and attendant, having no concern as to the means and methods of treatment, the recovery is far more certain.

The persons who are unable to repose confidence in any form of treatment except for a brief time, and who are suspicious and egotistical and determined to trust their own judgment, and insist upon deciding questions of treatment, make up very largely the incurable class. This exaltation and delusional state is often a symptom of insanity, and not infrequently represents the first stage of general paresis. Such patients dread control and are averse to following the uniform line of conduct planned by the physician. They insist on freedom to go and come and implicit trust in their promises to carry out the treatment. These need, first of all, sharp restraint, with full control of the surroundings, and absolute conformity to all rules and regulations. Without this, successful treatment is always difficult.

To those who have confidence in the means employed and show a disposition to trust implicitly to the physician and attendant, restraint of this kind is not necessary. Where they are willing to bear pain and discomfort and to make an effort to help themselves, recovery is rapid. The question of restraint is dependent largely on the condition of the individual; in some instances it is stimulating and helpful, while in others it is irritating and depressing. In all cases a certain amount of espionage and control is absolutely necessary. This can be ascertained only after personal observation and some study of the case. In some instances the surroundings of an institution and the fact of being at an asylum are drawbacks fully recognized, while in others the opposite condition obtains. To many, the personality of the physician or attendant and the evidence that they are in control are sufficiently stimulating, and persons are able to recover without further hinderance.

Surveillance should be continued for a long time after active

treatment has ceased, and the patient's condition and surroundings should be special subjects of inquiry for the purpose of shielding from temptation and from causes which favor relapse. Thus, the business or professional man should not go back at once to his old life and surroundings and subject himself to all the strains and drains which brought on his former condition. The effort of the physician should be to impress on the patient's mind the need of a radical change in his manner of living, and this should be done at the beginning of the treatment.

The profound neurasthenia associated with mental enfeeblement and moral palsies are conditions present in all cases. These facts should be considered in the treatment. The withdrawal of the drug removes an active cause, and is only a preliminary step. In many instances it simply unmasks conditions not suspected before, and at all times it enables the physician to lay down some successful plan of treatment for future restoration.

In the removal of the morphine three methods have warm advocates: first, the immediate and entire withdrawal; second, the rapid reduction extending over two or three days; third, the gradual reduction lasting two or three weeks. The first method, immediate withdrawal, has many advocates abroad. Levinstein practised it with success and urged it as the most rational procedure. The patients were shut up in an asylum and the morphine was withdrawn at once; bromides, hot baths, and hot soups were given freely. After the third day the withdrawal symptoms relaxed, and in a week the patient was quiet and comfortable. This method has been opposed and pronounced inhuman. Practically it is used in station-houses and jails where morphinists arrested for crime are forced to abandon the drug. The withdrawal symptoms often are not recognized as such, but are ascribed to some other condition. Many persons confined in jails have periods of acute illness due to this cause, from which they recover. It is exceedingly doubtful if the collapse from sudden withdrawal ever ends in death, although the effect upon the patient's mind and body is often very severe. In large cities physicians to the station-houses find laudanum and morphine most excellent remedies, particularly in the sudden collapse which indicates the strong probability of morphinism. In private practice this method is impracticable, although it has been tried with the consent of the patient. It

requires excellent surroundings and careful attendants as well as close medical watching.

The rapid reduction of the drug, covering a period of two or three days or longer, is feasible and very successful in many cases. It requires special surroundings, with trained help and careful medication. The usual course is to reduce the customary daily dose—no matter how large—to four or five grains; this is done within a day or two, and without much suffering, proving that the enormous doses used have not had their proportional effects. A large quantity of the morphine is doubtless unabsorbed, and remains in the system with the possibility of suddenly developing profound narcotism and death. Thus, a person using twenty grains daily will die suddenly from no observable cause; morphine in large doses has been taken for a long time without unusual symptoms or premonitions of death, when suddenly its cumulative action concentrates on the nerve centres and death follows. This possibility is always present, even when small doses are used, and then in most instances death is attributed to other causes.

The morphine can be discontinued easily if the doses are divided and given at short intervals, reducing the total quantity at the same time. Thus, a patient taking fifteen grains a day may not notice the change to five or six grains. It is essential in this rapid reduction to clear out the alimentary canal with salines or copious draughts of hot water; sometimes a calomel cathartic is very good. Soda preparations are very useful, even when relaxation of the bowels takes place. An interval of from twenty-four to forty-eight hours should elapse after the first withdrawal before another reduction is made. Usually one or two grains can be withheld, but the amount should be determined by the condition of the patient; and if the remainder is given at night, the withdrawal symptoms are less severe. Placeboes may be given if the mind is morbidly sensitive, but they should be nothing more than bitter tonics.

In this rapid withdrawal stage the physician should be careful not to use other narcotics as substitutes too early in the treatment. If while giving four grains of morphine a day cannabis indica or any of the bromides is given, the effects both of the morphine and the substitute will be uncertain. A fact to be remembered is that in opium cases narcotics neutralize each other's effects rather than

intensify them. Thus, morphine and hyoscyamus given together are antagonistic; either of these drugs alone would have a more decisive action than when combined. The bromides also work in the same way. Larger doses are required to produce bromism when morphine is used at the same time, and its cumulative action is more severe and long-continued. The same is noticeable in other drugs. Practically it is found better to abandon the morphine before the substitutes are used. Narcotics may be taken in the morning where the morphine has been taken the night before; in this rapid withdrawal it is customary to give the morphine at night and to use the substitute during the day. Tinctures of other drugs should not be used at this time, because of the danger of alcoholic narcotism. Certain persons are very susceptible to the paralyzing action of alcohol at this period.

Some of the remarkable cases reported of the painless withdrawal of morphine have been effected by simply substituting some tinctures for the alkaloid. The withdrawal of the morphine and the substitution of codeia and other alkaloids, or laudanum or other preparations of opium, is but trading one habit for another. Many of the specific preparations contain some form of opium, the substitution of which for morphine is simply a change in the form of the drug. To abandon morphine and to depend upon alcohol in its various forms is not curative in any sense, and no alcohol should be used in the withdrawal stage.

The acuteness of the insomnia, depression, and neuralgia which follow the rapid removal of morphine should be treated by baths, hot and cold water applications, and massage. When the alkaloid is entirely withdrawn, many drugs may be used to lessen the acuteness of the symptoms, among which are valerian, asafetida, hyoscyamine, cannabis indica, and some coal-tar derivatives. As a rule, they should be given in large doses, frequently repeated until several doses are taken, then abandoned. No one drug should be given more than two or three days at a time unless its effects are so marked as to demand its continuance. The vegetable narcotics seem to be valuable in many cases, but do not all act alike. In some cases they are very powerful; in others they are of no value. Phosphate of sodium is a valuable remedy, and can be used continuously during this period.

The rapid-withdrawal stage should not last more than six to

ten days, while in some instances a much shorter time is practicable. The reduction of the morphine to four or five grains the first day, and the third day its still further reduction to three grains taken at night, will be found most practicable. Then, if possible, substitute deodorized tincture of opium, in proportionate quantities, the fourth night. The sixth night this can be reduced still further, and then the morphine can be abandoned on the eighth or ninth day. After this time narcotics which have been found effective are to be given at night. These can be abandoned after one or two weeks without special suffering. Strychnine, quinine, and other active tonics are very valuable at this period. Faradism, massage, and confinement in bed, all have good effects.

This method of rapid withdrawal will tax the therapeutic resources and skill of the physician to the utmost. Each case will vary widely in both physical and psychical symptoms. In one instance water in the form of baths, hot applications, or spongings of the body will do well. In another, feeding, confinement in bed, and personal attention by attendants will be sufficient. In a third case exercise, mental diversions, and frequent change will lessen the intensity of the symptoms. In others drug restraint and narcotics are demanded imperatively. The same diversity of symptoms will appear after the morphine is withdrawn, and the same skill will be required to adapt special means to the desired end.

In the third method of treatment, the gradual reduction extending over a period of several weeks, much the same course will be pursued, only less rapidly. The morphine should be reduced to four or five grains during the first few days of treatment. Then a slow withdrawal daily or weekly should follow. Where the needle has been used, the difficulties will be increased because of the fascination which follows from the effects of drugs taken in this way. The needle should be abandoned as soon as possible and the drug be taken by the mouth. I have found solid opium to be better borne by the stomach than morphine. This, with the deodorized tincture, can be given in decreasing doses with good effect. This form of opium can be given concealed in bitter tonics, and, where the stomach will tolerate it, it is valuable as a substitute, and can be reduced in strength without being recognized by the patient. In many cases it is practicable to abandon the morphine for this form of drug almost immediately, and then slowly or rapidly to take

this away. Opium in the gum or powder form is often more efficacious as a substitute for morphine, the narcotism following it being more prolonged or agreeable because of the absence of stimulation; the withdrawal is less difficult and the symptoms have less of the mental and hysterical element.

Where the reduction period is likely to extend over several weeks, owing to the hypersensitiveness of the patient and his disinclination to bear pain and discomfort, great attention should be given to the diet and regular habits of living, avoiding all excesses and extremes of exercise, nervous excitement, and overeating. It is important to increase the vigor and strength of the patient in every possible way, as it is found that with increasing vigor the neuralgias disappear. Iron and phosphorus tonics are often very valuable. The salines in some form are indispensable, and the flushing of the alimentary canal by cathartics is equally important. Narcotics, as before remarked, are of little value except in the very last stages, when the opium is finally withdrawn. The best plan contemplates a gradual system of developing the vigor and healthy functional activity of the body and at the same time slowly removing the morphine.

In some instances the morphine has been reduced by infinitesimal fractions of a grain daily, on the supposition that nature would accommodate itself to this slow withdrawal. Others substitute some mild narcotic during the withdrawal process, but this, in the author's experience, has been very unsatisfactory. The exact plan and method of withdrawal must vary with the patient and the physician. Sometimes the surroundings have much influence, and in an institution where these can be controlled, mathematical exactness in the conditions may be followed out. The preferable plan is to drop the morphine in quarter- or half-grain doses at intervals of ten days or two weeks, and to accustom the system to adapt itself to the reduced doses by continuing daily a fixed amount. In one case a half grain was taken away every two weeks until only a half grain was used daily. The intervals between doses after the first few days were passed without much suffering. At the last the half grain was removed and bromides substituted for it; the second day hyoscyamine and trional were used with good effect. In a week or so the patient was able to do without any narcotic.

After the morphine is withdrawn, the severity of the irritation

and delirium is sometimes best relieved after the second day by return to the drug again in some concealed form for one or two doses. An example of this was that of a morphinist who, after the final withdrawal, was intensely melancholic and delusional. This condition increased until, on the evening of the second day, a dose of morphine concealed was given. The relief and sleep which followed lasted twenty-four hours, after which substitutes were able to produce a degree of comfort, and the restoration was rapid and uneventful. This course is not always followed by the same results. The patient will demand the same drug, not knowing what it is, and the skill of the physician will be taxed to find a substitute which will be satisfactory. Manias following the withdrawal of morphine can be broken up in this way, and also phobias, but great skill is necessary to prevent their recurrence. In one case of destructive mania from the withdrawal of morphine, the drug was given, again breaking up the mania; then this drug was substituted by forced cold and hot showers, which prevented the return of the mental disturbances, and final recovery ensued. After the crisis is past, the former substitutes may be given with excellent effects. If the patient's mind retains consciousness of the conditions which have existed, this treatment is followed by renewed confidence and faith of recovery.

Insomnia should not, as a rule, be treated by hypnotics. The danger of contracting another habit is so great that it is unwise to use any one of the hypnotics except for a brief time. Tobacco should be stopped early in the withdrawal treatment, as it always introduces serious complications, and after the withdrawal symptoms are passed its resumption is very commonly followed by relapse. Beef tea and beef extracts are unsatisfactory, and in most cases are nerve stimulants of decided inferiority, and seriously complicate the progress of the case. Fruit juices and grain products, with milk, are the best nutrients which can be given. Often an abdominal bandage, wet either in cold or hot water, has soothing effect on the sympathetic nerves of the abdomen, checking diarrhœa and gastric trouble. Cold water applications to the spine in the form of ice-bags are very serviceable.

The procedure which has been found most practical by the author is that of gradual reduction, going from stage to stage,—now slow, then rapid,—being governed by the history and present

condition of the case. It may be divided into three stages: First, the preparatory stage, in which an effort is made to ascertain the smallest amount of morphine which can be taken without discomfort to the patient. This sometimes requires an extended observation of a week or more. Many patients use far more morphine than they imagine, being careless and inaccurate as to the time when taken and quantity used. Others intentionally deceive themselves and others, boasting that they only take a certain amount, when in reality this is only a minimum. Having secured the proper surroundings and control of the case, the patient's statement of the amount he is taking is accepted, and he is given a like amount for the purpose of testing his accuracy. If this is found to produce marked narcosis, it is evident that it is more than is essential for comfort. If he is restless and uneasy, it is less than his usual dose. These conditions will vary largely the first two or three days. The fact of coming under treatment in strange surroundings and under new conditions furnishes a psychical element which will derange the nervous system, requiring more than the accustomed dose of morphine at first. After the patient has acquired a degree of confidence and become used to the surroundings, an approximate average state can be attained. If it is found that he is comfortable on eight or ten grains a day, this is assumed to be the average quantity necessary to produce reasonable sedation.

Having ascertained the character of the case, the next question is the gradual or rapid withdrawal of the drug. If the habit is an acquired one in a person previously well and free from neurotic strain and organic disease, is associated with the use of spirits, and the duration of addiction is two or three years, a rapid withdrawal is the most practicable. The conditions present are always neurasthenia and anæmia, and various functional disturbances which are made worse by concealment and the narcotism of morphine. Having found the amount of morphine the person takes, and corrected in a measure the digestive disturbances which exist, the second stage of treatment begins. The first step will be to abandon the morning dose of morphine and to concentrate the amount given from noon to six in the afternoon, rarely giving any after six or seven o'clock in the evening, the object being to get the narcotic effect during the night and to break up the plan of the previous use. Where it had been taken in small doses at short intervals, larger

doses are given at longer intervals. If the absence of morphine in the early morning causes suffering, baths are given. Stimulating foods and hot milk or acid drinks are administered if the stomach will bear them. If the discomfort is severe, opium pills of half a grain or one grain may be given. When the system is accustomed to this change the morphine may be reduced one-half the usual dose at once. If ten grains are taken daily, five grains will suffice. If given by the needle, the diminished amount is seldom recognized. If by the stomach, the effects are more apparent. Later, according to the condition of the patient, a still further diminution is made, and if the suffering is marked there is given a preparation of cinchona bark, usually the infusion, in drachm doses, combined with ten drops of deodorized tincture of opium. Most cases bear reduction without any particular discomfort down to one or two grains. Then increasing doses of tincture of opium with bark becomes a good substitute, and the morphine can be withdrawn at once. If the needle habit has been acquired, the use of the needle must be kept up with regularity, occasionally substituting one or two grains of dionin in place of the morphine.

When opium is not borne well by the stomach, pills of lupulin, black haw, valerian, cannabis indica, and hyoscyamus may be alternated until some one is found having narcotic properties sufficient to lessen the extreme irritation. Baths are to be given every day during the reduction period, and phosphate of sodium in small doses should be given two or three times a day. In from ten to twenty days the morphine can be entirely abandoned.

Then comes the third stage of treatment for the withdrawal symptoms. The effort here will be to diminish their intensity, particularly that of insomnia and restlessness. Prolonged hot baths, either in a tub or from a shower, or from packs, are very useful, and very satisfactory in many cases. Nitrate of strychnine is in some cases of great service, particularly where the needle is used. Beginning with one-thirtieth of a grain, the amount may be increased to one-tenth of a grain given three or four times a day. If it produces excitement, both muscular and mental, it should be discontinued. Phosphoric acid with nux vomica are followed by pronounced effects. Extract of horse nettle, given in fifteen- or twenty-drop doses, has been found very efficacious in removing the restlessness and encouraging sleep during this stage. Care should

be taken of the diet. Small quantities of food should be taken at short intervals rather than full meals at stated hours. If the bowels become troublesome in the dysenteric discharges, increased doses of cinchona with capsicum will be useful. Tea and coffee during this period have been in some instances medicinal in calming the restlessness and allaying the discomforts. In others, they are both stimulating and irritating. Cocoa seems preferable, and should be used often, very hot. Electricity has been found useful in some cases, while in others it is an irritant. The static current seems best adapted to the conditions, and is often followed by decided rest and relief from discomfort. Electrical baths are very highly valued by some authorities, but it is probable that their value depends upon the peculiarities of the case. The electric light bath has been used recently with the best results and promises to be a most valuable remedy. The patient is placed in an electric room and after free perspiration of a few moments is put under a shower bath and rubbed carefully; he then rests in bed for an hour or more. The effect is very restful and sleep follows in all cases. Experience indicates that this treatment is not always practicable, and cannot be used as a general remedy.

The withdrawal period may last from four to ten days, sometimes longer. When it has passed away, the patient recovers in a large measure, and only suffers from general weakness and depression of spirits. Care should be taken by this time not to use any drugs that are known to the patient, particularly those that are likely to produce pleasing effects and be taken afterwards for their quieting influence. The muscular delirium, or intense desire to use the muscles of the legs in walking, is overcome by massage and vibratory machines that shake and push the muscles by force. A few minutes of this exercise seems to expend the muscular energy and to take away this uneasy feeling. This peculiar delirium cannot be overcome by exercise in the open air without danger of reaction. In one case, walks of ten or twelve miles a day for a time resulted in extreme prostration and relapse; in another case excessive exercise in a gymnasium was followed by the same result. Limited indulgence in this desire is helpful, with massage and hot and cold baths daily. Warm baths at night, if not too stimulating, followed by rubbing, seem to be more sedative than in the early stages of treatment. Mild exercise in the open air is often of great

value, the difficulty being that the person is liable to over-exert himself and suffer from muscular fatigue. Short rides and walks are of more advantage, the intervals being passed in a reclining position on a couch. In most cases, a most excellent plan is to remove the clothes in the middle of the day, darken the windows, and go to bed with all the conditions favorable for sleep. After a short time, lengthening periods of sleep will follow. During the withdrawal period the patient should see very little company, attend to no business, and have no strain on the mind if it can possibly be avoided. In some cases, particularly in those who are over-fed and inclined to corpulency, a Turkish bath with mild rubbing every day, either in the forenoon or early in the evening, will be found very advantageous. In a thin, spare person, with acute sensitiveness and hyperæsthesia of the skin, showers and hand-massage are of great value. In the absence of these measures, sponging with warm salt water or with water containing four ounces of vinegar to the gallon, is very soothing. For the various anæsthetic and hyperæsthetic conditions, also for the local neuralgias, hot and cold applications are very useful.

THE DIAGNOSIS AND TREATMENT OF OSTEOMYELITIS.

CLINICAL LECTURE DELIVERED AT THE NECKER HOSPITAL.

BY P. MAUCLAIRE, M.D.,

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Gentlemen,—I propose to take advantage of the presence in our wards of several patients suffering from osteomyelitis to speak about the diagnosis and treatment of the different forms of this infection, to which every one is exposed until he reaches the end of his period of growth.

The first patient is a youth aged fifteen, who on July 2 first experienced pain in the upper part of the tibia. Little by little this increased, until on the 5th he entered the hospital in a general state resembling that of typhoid fever, with headache, a temperature of 102° F., and absolute functional impotence of the limb. There was swelling accompanied by pain, redness, and heat, especially marked at the tibial spina. At this point examination revealed a collection of pus, and we found the case to be one of subperiosteal abscess situated over the junction-cartilage of the upper part of the tibia, and symptomatic of osteomyelitis of a bone in course of development. We immediately performed trephining of the infected medullary cavity, and provided drainage for it. The patient then developed sero-purulent arthritis,—a frequent complication,—which was treated and cured by simple aspiration.

At the present time this patient's condition is satisfactory, the joint being normal, though there still remains a small fistula of the bone which will probably soon heal up; but we must not forget that he may possibly have a recurrence of the disorder, and that another attack of osteomyelitis may take place either in the same or in some other bone.

The second patient is a young woman aged eighteen, who entered our wards suffering from an ordinary attack of appendicitis, with pain at McBurney's point, peritoneal reaction, muscular contrac-

tion, fever, vomiting, etc. A few days after the appearance of this appendicular crisis she manifested symptoms of recurrence of an attack of osteomyelitis of the tibia, from which she had suffered several years previously. The disorder had been treated by central trephining of the bone, and there remained a large excavation of the tibia. In spite of careful searching and the use of the curette, I was not able to find a sequestrum. This coincidence of an attack of appendicitis followed by a recurrence of osteomyelitis is very interesting from a point of view of general pathology. Which was the causative lesion?

The third patient is a man aged thirty-seven who towards the end of August first noticed severe pain in the left knee, followed by swelling of the inner side of the upper portion of the tibia. Walking soon became impossible, and on May 15 the swelling of the tibia gave, on palpation, somewhat the sensation of snow. There were heat and pain, well localized to the upper epiphysis of the left tibia, and at the same time the patient complained of headache and prostration, while the temperature oscillated between 101° and 103° F. Trephining, performed on May 20, showed the bone to be slightly thickened, pale, and fatty, somewhat resembling gumma tissue. A drainage-tube was inserted in the wound and the entire limb placed in an apparatus. Suppuration lasted four months, at the end of which period the patient was able to walk.

This man has, in addition, a characteristic deformity of the bones of the nose, which, together with his age, leads to the conclusion that the disorder is of a specific nature. The patient denies having had syphilis, and we were unable to put him on the specific treatment, which would probably have cured him in a short time. The deformity of the bones of the nose is quite typical, but the patient said it was due to an accident, although he is unable to give us any details concerning it. We could not find any trace of syphilis elsewhere.

The fourth patient is a very interesting case. This woman had never been ill up to the age of twenty-two. At that time symptoms of infection of the bones of the left elbow appeared, followed by similar manifestations in the diaphyses of the two tibias. The case was at first thought to be an ordinary one of osteomyelitis, and the infected bones were trephined in a hospital in the country; this operation did not, however, impede the evo-

lution of the disorder. The point that most attracted attention was the peculiar shape of the tibial diaphyses, which were thickened and somewhat convex to the front; the thickening was especially manifest on the inner side of the bone, though the total size of the latter had increased. Again, fistulæ from the bone gave issue to a sort of gummy serum quite characteristic. For these reasons I rejected the hypothesis of infection by the staphylococcus, and made the diagnosis of acquired syphilis of the bones. The patient was immediately put on specific treatment at high pressure (deep injections of gray oil, and iodide by the mouth). The disorder yielded little by little; but at the end of six weeks the result is very satisfactory and fully confirms the diagnosis. The reason why recovery was somewhat slow is that the syphilitic infection was complicated by the ordinary suppurative process of the bone.

In addition to the foregoing cases I have several others—little patients taken from a neighboring children's ward—who have suffered from osteomyelitis of the femur, tibia, humerus, and lower jaw; so that you will have had before you a number of types of patients by which you can be guided in making the diagnosis of infectious disorders of the bones.

Let us now determine the mode of procedure by which to distinguish the different forms of infection of the bones. I will assume that your case is in reality one of bony infection, and that all the eruptive fevers have been eliminated, particularly typhoid fever. I have many times been called on to certify the latter diagnosis, and have found a subperiosteal abscess that had passed unnoticed.

The first point to settle is whether the case is not one of tuberculosis of the bone, a very common disorder. In the majority of cases but little trouble will be experienced in coming to a conclusion on this point. In the first place you will be guided by the seat of the lesion; in osteomyelitis the disorder is localized at the level of the junction-cartilage, usually respects the neighboring joint, and has a tendency to spread towards the diaphysis of the bone. Tuberculosis, on the contrary, invades the joint in the form of a white swelling, and limits itself to the epiphysis; it is very unusual for it to extend to the diaphyses. The difference is, therefore, very distinct, and the only exception is that of an unusual hybrid form which has been named osteomyelitic osteo-arthritis.

In the second place, tuberculosis of the bones is usually accompanied by other bacillary manifestations at different points of the body, by a bad general condition, and particularly by tubercular adenitis. In addition to this, the evolution of tuberculosis is much slower and more chronic than that of osteomyelitis, which is essentially acute,—in some instances extremely so; whence the different terms by which it has been called (typhus of the limbs, deep abscess, diffuse suppurative periostitis, etc.), and which well characterize the clinical evolution of the infection due to the staphylococcus aureus. Still, there are cases in which the diagnosis is not easy, and there is at the present time in our wards a patient showing a number of fistulæ of the femur, in whom the diagnosis of tuberculosis was made positive only by the recent appearance of adenitis of the neck.

When tuberculosis has been eliminated, it will be necessary to ascertain whether the case is not one of syphilis of the bones, which may be either hereditary or acquired. In the hereditary forms of syphilis the disorder generally appears in the tibia, which shows hypertrophy of a very peculiar kind; this will generally be found on the inner side of the bone, which will be larger than usual and abnormally irregular, while the whole bone will show an antero-posterior curve making the diaphysis look somewhat like a sabre. This deformity is different from that met with in many cases of rhachitis, in which the tibia is flattened, the transverse diameter of the diaphysis is lessened, and the inner side of the bone is not uneven. Besides this, these patients show the usual lesions of the ribs, etc. You must also, in cases in which hereditary syphilis of the bones is suspected, examine the clavicles and the edges of the ulnar bones, as at these points additional characteristic deformities will frequently be detected.

Attention must likewise be called to a false form of infantile paralysis, described by Parrot as a variety of congenital infection of the bones of a syphilitic nature. This disorder is characterized by pain in the shoulder or knee, accompanied sometimes by partial dislocation of the joint; there are some signs of inflammation, together with impotence of the limb. These symptoms are due to dislocation of the epiphysis, caused by the development of syphilitic gummata in the junction-cartilages.

Finally, you must search carefully for the usual stigmata of

hereditary syphilis: Hutchinson's signs—sunken nose, deformed teeth, interstitial keratitis, deafness, and pointed arch of the palate. This examination must be minute and systematic, whatever the social situation of the patient; since syphilis respects neither person nor rank.

In acquired syphilis of the bones, a patient, at variable periods of time after the appearance of the chancre, complains of pain on the inner side of the tibia, and examination of that region will disclose a swelling characteristic of periostitis due to gummata. In other cases the lesion is a central one, or osteomyelitis of the tibia, due to the same cause. In either case this disorder can easily be differentiated from osteomyelitis of the period of growth if one be guided chiefly by its seat and evolution; since, in opposition to what occurs in osteomyelitis, which appears near the junction-cartilage, the syphilitic gumma invades the diaphysis in its entirety, as was the case with our fourth patient. Again, the former disease is usually acute, whereas the latter is essentially a chronic disorder.

In this way you can eliminate the two most important disorders of the bones with which a case of osteomyelitis of the period of growth could be confounded: syphilis and tuberculosis. You must now endeavor to settle the bacteriological variety with which you have to deal, since osteomyelitis of the period of growth may be due to different pathogenic agents, and M. Lannelongue has shown that each microbian species corresponds to a special clinical symptomatology. Let me mention these different bacteriological varieties.

In most cases the disease is caused by the *Staphylococcus aureus;* this is the common form, whether acute, prolonged, or chronic.

The white staphylococcus corresponds to a subacute form of evolution.

Certain cases of osteomyelitis in newly-born babes are caused by the streptococcus, the infection being transmitted from the mother suffering from puerperal fever to the child by the vessels of the umbilical cord. This variety is said to be less destructive than those due to staphylococci, and to be characterized particularly by the frequency of suppurative arthritis.

There is a form of osteomyelitis, due to the pneumococcus, which occurs during an attack of pneumonia; its evolution is sub-

acute, and as a general thing it is not accompanied by the formation of a sequestrum.

Finally, cases of infection of the bones by Eberth's bacillus have been reported, occurring during convalescence from typhoid fever. This form of osteomyelitis is usually found in the tibia, and its peculiarity is that it is almost always superficial. In short, it is an osteoperiostitis rather than true osteomyelitis.

Forms due to the gonococcus have also been described, in which the microbe of that disorder has been found.

Attempts have been made to divide cases of osteomyelitis into two groups, from an anatomical point of view: subperiosteal and central forms. Lannelongue has called attention to the fact that the lesion generally begins at the centre of the bone and thence extends to its superficial portions; consequently, the varieties that are supposed to be subperiosteal appear to me to need further study.

In addition to these distinctions based on the nature of the infectious agent or on the chief seat of its lesions, the clinical varieties of osteomyelitis must be considered, and I will briefly refer to them.

In the first place, there is an acute form, characterized by sudden swelling of the bone and by a very serious general condition (typhus of the limbs), which is sometimes excessively acute (diffuse suppurative periostitis) and of which the diagnosis is easy. The localized pain draws immediate attention to the diseased portion of the bone; but in certain cases it is possible to mistake the lesion of the bone, and to diagnose lymphangitis, or a simple circumscribed abscess of the subcutaneous or deeper cellular tissue, or a diffuse form of suppuration. For this reason, and particularly with children, the possibility of osteomyelitis must always be borne in mind. If you do not find a wound or some sort of lesion on the surface to account for the infection and the swelling of the bone, do not rest satisfied with superficial palpation, but go in deeper and, if it be supposed that the swelling has occurred on the leg, look for the pain at the junction-cartilages of the tibia, and in many cases its ascertainment by touch will enable you to make a certain diagnosis of osteomyelitis of the period of growth. On the other hand, be very suspicious of what are called "growing pains;" growth ought to occur without pain, and these are usually nothing but light cases of osteomyelitis.

In opposition to the acute form, but in intimate pathogenic connection with it, should be placed what is known as prolonged osteomyelitis, which is characterized by a first acute attack, similar to what has just been described, but followed at a greater or less interval by reappearance of the same symptoms. Such patients have during their period of growth an evident attack characterized by fever, pain localized to the junction-cartilage, with or without subperiosteal abscess and elimination of a sequestrum, spontaneous or otherwise; after which they apparently recover. But after several years (ten, twenty, or more) a swelling similar to the first appears in some other portion of the bone that was formerly affected. In some this swelling is caused by an abscess of the bone, with the excruciating pain which is so characteristic; in others it is a simple hyperostosis. These tardy recurrences may even be repeated, and this without regard to age, as Lannelongue quotes a case of prolonged osteomyelitis in a man aged seventy. You will easily recognize this form of infection by its seat, history, and other characteristics, and will be able to differentiate it from tuberculosis, osteoperiostitis whether rheumatic or otherwise, exostosis of the period of growth, and osteosarcoma.

A third clinical variety of osteomyelitis is chronic from the outset. In this form there is no initial acute attack; but during or after the period of growth a swelling of the bone is observed which can be distinguished by its seat from a tuberculous lesion, but which in some cases can be differentiated from an osteosarcoma only by an exploratory incision.

Having made the diagnosis of osteomyelitis, you have still to determine whether or not there is a central sequestrum. The latter exists in nine cases out of ten, but in the prolonged or chronic variety it sometimes eludes detection on account of the thickness of the bony tissue by which it is surrounded; these hyperostoses are traversed in places by fistulæ which lead towards the sequestrum, loose in a cavity or still adherent and incrusted.

The presence of such infected sequestra make the prognosis more sombre, for osteomyelitis is of itself a very serious disorder, and still more so when there is an infected sequestrum which does not come away; the bone remains fistulous for an indefinite period if not operated upon, and these chronic fistulæ may become complicated with lymphangitis, erysipelas, tuberculosis, and epithelioma.

The nature of the infectious agent likewise plays a certain part in the progress of the disease. I called attention to the relative benignity of cases due to Eberth's bacillus, and you will remember that the most serious forms are those due to the *Staphylococcus aureus*. A bacteriological examination, therefore, will guide you in settling the prognosis of a given case.

Another point showing the seriousness of this disease, in addition to the intensity and gravity of the acute manifestations of septicæmia by which it is accompanied, is to be found in the lasting deformities that it may cause. An attack of this sort sometimes results in one of the patient's legs becoming shorter than the other, or else in articular changes very like those of hip disease; partial dislocation of the tibia occurs, with spontaneous fractures, separation of the epiphyses, and partial destruction of the diseased bone.

Pseudarthrosis, bony atrophy by destruction of junction-cartilages, checked growth, various inflections, and articular dislocation are also sometimes met with.

I have now a few words to say concerning the treatment, which I shall endeavor to make very clear. In the acute form the treatment is very simple, and may be summed up as follows: incision of the subperiosteal abscess and disinfection of the centre of the bone by trephining and drainage of the bony focus and of the infected soft tissues.

Early resection of the bone, which was advised to avoid the formation of sequestra, has not given the results that were expected, as these sequestra are too extended from the outset; it should be noted that at present early trephining and methodical drainage of the soft tissues, and especially of the joints, have rendered amputation very rarely necessary.

In fistulous, chronic osteomyelitis the bone must be trephined and the central cavity searched to find the septic sequestrum, following the fistulæ when possible, and going through the thickening of the periosteum as well as the shell that may be about the sequestrum. Care must be taken not to wound the vessels of the region. This emptying of the bone is not without its drawbacks, and attempts have been made to fill the breach by grafting either living or decalcified bone. The results of this grafting are not constant, owing to the fact that complete and preliminary disinfection of the bony cavity is difficult. The same can be said of the use of iodo-

form gauze. A number of osteoplastic attempts have been made, and I also have had recourse to this plan. The method consists in cutting one of the bony flaps resulting from excavating the diaphysis, keeping the skin that covers it, and turning down this osteocutaneous flap over the cavity. I think that in this way I have in several cases hastened the healing of a cavity left after removing an infected sequestrum.

Attempts have also been made to separate the skin on each side of the excavation and to draw it over into the cavity made in the bone. I obtained one excellent result from this method.

Finally, some surgeons have endeavored, after thorough disinfection of the bony cavity by means of boiling oil heated with the actual cautery, or of hydrogen peroxide, to perform obturation by means of a sort of filling similar to those used by dentists. Many researches will no doubt be made in this direction.

The orthopædic deformities left by these attacks must be remedied either by section, by breaking of the bone, by resection, or as a last resort by amputation.

THE URTICARIAS; THEIR CAUSES, VARIETIES, AND TREATMENT.

CLINICAL LECTURE DELIVERED AT THE ST. LOUIS HOSPITAL, PARIS, FRANCE.

BY PROFESSOR H. HALLOPEAU,

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GENTLEMEN,—Urticaria (or, as we prefer to call it in France, the urticarias) constitutes an extremely interesting group of skin diseases. This interest has not decreased of late years, but has rather increased, since we have come to realize the importance of the nervous system in producing lesions of the skin, and the many forms of urticarias that may occur. The departure from the simplicity of former times in the matter of diet has given rise to a greater number of cases of urticaria than the older physicians were accustomed to see. The introduction of certain new remedies,—notably the coal-tar products,—that have been of so much service in medicine but have at times given rise to extremely inconvenient by-symptoms, and the use of certain biological products, as for instance the antitoxic serums, also have made the opportunity for the observation of urticaria more common than it was. Despite the opportunities provided for varied experience and observation, however, the problems involved in the disease have not all been solved, which is probably another reason for special interest in it, since mysteries always attract attention.

One of the most important phases in urticaria is undoubtedly the existence of a predisposition to the occurrence of serous infiltration of the cutaneous tissues. There are certain people who possess normally the quality known as autographism, or dermographism. In these persons the simplest form of irritation of the skin, as for instance a gentle stroke with the finger-nail or the drawing of a line lightly on it with a pencil, sometimes even the passing of a sponge over the surface, or the rubbing of the fold of a garment, will produce a distinct linear infiltration of the tissues of the derma. I have shown you more than once individuals who were apparently

in perfectly normal health, in whom these slight irritations produced urticarial effects. This extreme of reaction is due either to an abnormal excitability of the walls of the blood-vessels in the skin, or to superirritability of the motor nerves to these blood-vessels.

Dermographism undoubtedly occurs most commonly among patients suffering from some general nervous condition. A number of French authorities have called attention to the fact that the occurrence of dermographism among hysterical patients in olden times was without doubt ascribed to demoniac influence. The marks produced on them were set down as due to compact with the devil or to some other equally infamous condition, and more than once poor, unoffending sufferers from major hysteria have been put to death as witches or sorcerers because of this tendency of their skin to react to slight irritation. Neurotic individuals are not, however, the only subjects of the symptoms, for dermographism has been observed among persons absolutely free from any other nervous trouble.

By urticaria we usually mean an eruption composed of limited elevations of the cutaneous surface, always of irregular outline but distinctly circumscribed; these eruptive lesions are whitish or slightly pink in color. They are of rather firm consistency, and have something of an elastic feel,—that is, they yield slightly on pressure but return to their former size at once. The separate lesions may be produced and disappear within a few minutes, and it is somewhat of an exception to have urticarial lesions last for some hours. It is not difficult to see that they are due to a limited effusion of serum into the skin; this circumscribed œdema constitutes the essence of the disease. Urticarial eruptions are usually accompanied by a special sensation of itching or burning, but may occur without any sensation; in this respect, though the etymology of the name involves the idea of itchiness, the urticarial character of the disease is not essential.

Perhaps the best explanation of the origin of urticaria is that it is an exaggeration of the vasomotor disturbance of the skin tissues that occurs almost normally in some individuals after very slight irritation. You are all familiar with the red line that follows the drawing of the finger-nail across the skin of patients who are suffering from meningitis. This has been called the *tache cérébrale,* and

is evidently due to the fact that the central nervous system, owing to the pressure upon it, has lost its usual control over the vasomotor nerves to the minute blood-vessels of the skin. Sometimes the line is only a reddish mark, and no furrow or welt occurs, but in general there is a distinct elevation of the cutaneous tissue along the line. The elevation of the skin is very marked in some cases, and spreads even beyond the portions of tissues directly irritated. Barthélemy has studied certain cases in which the elevation extended so widely that it could be compared to a goose-quill placed under the derma.

I made a curious observation regarding some of these cases of marked dermographism a few years ago,—that over the regions of urticarial irritation the temperature of the skin is elevated by a degree to a degree and a half above that of surrounding portions. This is probably due to the more active circulation in these parts, which can be seen by the greater redness of the skin, and it is likely that a corresponding elevation of temperature exists in all urticarial lesions, though I do not know that this has ever been demonstrated by actual observation.

As a rule the local irritation which gives rise to the urticarial lesions is of toxic origin. An excellent example of this is furnished by the lesions produced by contact with the so-called stinging-nettle, and by the bites of certain insects, notably the flea and the bedbug. The importance of the individual idiosyncrasy in the matter of urticaria is evidenced in the effects produced by these bites; many persons are scarcely affected at all, in some there is only the slightest amount of redness and irritation, while in others the bite produces not only an itching urticarial lesion but a slight hemorrhage under the skin. It has been observed also that individuals, after suffering for many years from urticaria provoked by the bites of insects, may take on a condition in which they suffer no reaction from these bites, perhaps because they have acquired immunity, or because the condition of the nervous system has changed and there is no nervous reaction in the vasomotor mechanism of the skin.

The most familiar form of urticaria is that which is seen in subjects predisposed to such lesions after they have eaten certain foods, —usually fish of some kind. Not all fish, however, have the same effect, even on the same person, and generally some special form is the cause. Shell-fish of various kinds—the mollusca and crustacea

in particular—are most frequently blamed; mussels have an especially bad reputation. Fresh-water fish seldom produce urticaria as a general rule, but almost any form of sea-fish may affect certain susceptible people. Among the meats it is mainly those that have been allowed to become gamy that are likely to produce urticarial lesions. Pork may have this effect when perfectly fresh; but generally it is thus active only in the form of sausage, when it may have been kept for some time and have begun to be gamy, its gaminess being concealed by various condiments that are added in the process of making the sausage. Besides the meats there are certain cheeses that are liable to be followed by urticaria in people who have an idiosyncrasy for the irritating products they contain; especially is this the case with cheeses that go through processes of fermentation and micro-organismal change during their manufacture. Since gamy meats have gone through processes analogous to the fermentation and micro-organismal changes in these cheeses, and since sea-fish (especially the various forms of shell-fish) are particularly liable to degenerative and almost putrefactive changes before they find their way to the table, it is not difficult to determine that it is probably these fermentative changes in food materials that give rise to the abnormal cutaneous irritability.

Among the fruits, strawberries, currants, and raspberries are most frequently blamed for the production of urticaria It is a noteworthy fact that people who are very susceptible to urticaria from eating strawberries may be able to eat raspberries with impunity, while to other patients raspberries are a frequent source of urticarial irritation. The idiosyncrasy may limit itself to currants alone, or even to one special form of currants to the exclusion of all other varieties.

Certain medicinal substances may have a similar effect, though the idiosyncrasies for drugs are fortunately very rare. Vin de quinquina, for instance, a remedy rather popular in France as an appetizing tonic, has been known to be followed by rather severe urticaria; other familiar drugs that have the same action are antipyrin, copaiba, turpentine, ordinary sulphate of quinine, salicylate of sodium, santonin, and iodide of potassium. It is a very curious thing that food materials and medicinal substances entirely inoffensive for the great majority of people should become so irritatingly toxic for a few individuals, and should invariably produce urticaria.

Certain pathogenic agents, when they become localized in the cutaneous tissues, can by special exception give rise to an urticarial eruption. I have seen a case in which the disease known as Gibert's pityriasis rubra was really a manifestation of this kind. The appearance of the urticaria does not seem due so much to the influence of the toxic agent as to the predisposition of the individual.

The preliminary disturbance of the system due to the presence of an infectious agent that is later to cause one of the specific exanthemata may at times be accompanied by an urticarial eruption. The smallpox eruption, for instance, may be preceded by a rather wide-spread urticaria, which has been seen quite frequently, especially of late years, as a systemic symptom of vaccination; measles and scarlet fever sometimes have urticaria as a preliminary stage. The disease has been known to occur during the course of a dysentery. Malarial urticaria, apart from the rare cases in which the eruption is due to the administration of quinine, has also been observed. There seems no reason to doubt that the toxins of the specific infectious disease are the cause of the urticaria in these cases.

The toxic action of certain products absorbed from the digestive tract during the course of chronic digestive disturbances may also give rise to urticaria. Fermentative dyspepsia, even when of so latent a character as to be almost unrecognizable by the ordinary signs of that disorder, may be a source. Icterus, with its accompaniment of irritating substances in the circulation, is often associated with a marked tendency to urticaria. It has often been noted that during the progress of a severe nephritis, especially when this is accompanied by albuminuria and by a dropsical condition, urticarial lesions are apt to be seen. Perhaps it is also for toxic reasons, rather than because of nervous irritation, that diseases of the uterus and its adnexa are accompanied by urticaria in certain cases, as these diseases are prone to cause disturbances of nutrition and digestion. Debove showed some years ago that the resorption of fluid from large serous cavities into which it has been effused may be followed by urticaria; he made actual observations with filtered fluid taken from hydatid cysts of the liver.

It is needless to say that in all these cases the lesions are produced only in people who are especially liable to the affection. It has been noted in some cases merely as the result of vivid emotion,

when it would seem that the nervous strain of itself—without the intervention of any toxic substance—caused the disturbance of the vasomotor mechanism of the capillary blood-vessels in the skin. It has been said that urticaria can be caused by suggestion, but this has not been proved, and the cases cited seem to us to be explicable on other grounds.

Urticaria is by no means necessarily limited to the external integument; certain acute and chronic forms of œdema probably affect the mucosa as well as the cutaneous tissues. Urticarial patches have been seen on the mucous membrane of the lips, the inside of the cheeks, over the tongue, on the veil of the palate, and in the pharynx. An urticaria of the larynx has been described, and it is thought that this can be a cause for the so-called œdema of the glottis,—the transudation of serum into the submucous tissues lying near the vocal cords. The existence of gastric and intestinal urticaria, and even of a bronchial form, seems probable; there may also be ovarian and uterine forms. Vomiting, diarrhœa, or dyspnœa are not unusual at the same time as the skin lesions of urticaria, and there are certain uterine disorders that appear also to point to a similar state of affairs, though there has been no direct observation in this matter.

There is a very interesting variety of urticaria which has lately attracted considerable attention, and has received a number of names,—acute œdema or, as it has been called by Quincke, angioneurotic œdema; English observers at times term it giant urticaria. The main feature of this disease is the sudden production of considerable swelling in one or more parts of the body, and sometimes in different parts at different times. These swellings are sometimes as large as the palm of the hand, and in rare cases may be even larger. The skin over these swellings is red and tensely stretched, and the edges of the swelling are somewhat raised above the surrounding surface; the tumefaction is rather soft in consistency. The most surprising feature of the disease is the absence of sensory disturbance. It is not accompanied by pain, nor is there usually any itching; it may occur entirely without the patient's knowledge, until some one calls his attention to it. At the end of some hours the swelling disappears, leaving no traces. Other attacks of the same kind may occur in other parts of the body. These symptoms continue to recur, with or without more or less prolonged intervals

of entire freedom, sometimes for weeks, or even for months; at times the history of such a case may extend over years. In spite of the absence of itching, the mode of development of these lesions, and the symptomatic picture they present, seem to indicate that they are closely related to chronic urticaria.

In chronic urticaria there is a continual recurrence of new attacks, which may come on every day, sometimes at a fixed hour, though usually at longer intervals. These attacks may occur during apparently good health, and I have seen them when there was not another morbid symptom in the case; yet they may last for months or even for years. Schütz attributes them to the influence of tactile impressions, or to sensations of heat or cold. Entrance into a warm room, the warmth of the bed-clothing, the cold air when the patient goes out, or cold applications of any kind, may bring them on. They may be prevented by exhausting the irritability of the vasomotor nerves; urticaria caused by cold, for instance, disappears when warm applications are made, and that which is produced by excess of heat may be relieved by applications of ice.

There are certain forms of chronic urticaria which come on in young children, especially in those who suffer from rhachitis, and which are undoubtedly connected with gastro-intestinal fermentative disturbances, as urticaria is so frequently in the adult. This form of urticaria has been studied particularly by English observers, and is sometimes known as lichen urticatus. The centre of the urticarial lesion is occupied by a papule at times, and sometimes by a vesicle or a pustule. Not infrequently this central portion corresponds to a sebaceous follicle. I myself have come to the conclusion that these lesions in children are due especially to scratching, and right here at St. Louis Hospital Brocq has shown that such lichenoid eruptions form one of the ways by which the skin reacts to irritation by scratching.

Besides these chronic forms, which deserve rather the name of relapsing urticaria, there are some urticarial lesions which, instead of disappearing at the end of a few hours, may last for many years, and the original swelling may be increased from time to time as the result of renewed attacks. The swelling is particularly noticeable because of its hardness and its resistance to the pressure of the finger; it recalls quite vividly the sensation produced by the palpation of myxœdematous tissue. The favorite seat for these swell-

ings is on the face, where they cause a persistent tumefaction of the eyelids, which often gives rise to considerable deformity of the countenance; the upper eyelids particularly are deformed, and the palpebral fissure is sometimes completely obliterated. The cheeks present a considerable elevation above the fold which separates them from the lobule of the nose, and the mouth-opening is liable to be retracted; a double chin also is not uncommon.

One of the most interesting forms of urticaria is that which is followed by pigmentation. We have simple pigmented urticaria, chronic urticaria with hemorrhagic pigmentation, lichenoid urticaria with subsequent persistent deposition of pigment, or erythematous urticaria with cyclical pigmentation. It has been noted that lichen urticatus may be followed by persistent splotches of pigmentation,—the so-called macular form of the disease. Pigmented urticaria is characterized by successive eruptions of erythematous elevated patches which correspond to the ordinary lesions of urticaria, but which, instead of disappearing after a short time, persist because of the pigment that is deposited in the tissues. The color of these lesions is usually about that of chamois skin; it resembles very closely that which is seen in patches of xanthelasma around the eyelids. In these cases relapsing attacks, which cause further deposition of pigment, are almost the rule, but the pigmented spots due to even a single attack may persist for a long time. This form of urticaria is likely to be accompanied by rather severe itching. As the result of this vesicles or even bullæ may appear. This affection is rare in the adult, but it does occur occasionally, and so cannot be considered as limited absolutely to the child, as has been thought.

A curious feature of this disease is that the eruption may occur in parallel bands, and have a zone-like distribution that reminds one somewhat of shingles, except that the form of the lesion is different. The lesions may leave indelible cicatrices. As a rule this form of urticaria is accompanied by the enlargement of neighboring lymph-nodes. Probably, as Besnier thinks, the disease is due to a trophoneurotic condition, but the enlargements of the lymphatics might lead one to think of infectious or at least irritant toxic material. Sometimes the lesions are only simple macules from the beginning, instead of occurring in raised patches. Little can be done for the disease in this form; it usually heals spontaneously after some years.

A number of mast-cells are found in the derma in this form of urticaria, which is the only one that gives any characteristic anatomical or histological picture by which it can be distinguished under the microscope from other forms of skin lesions. The mast-cells occur constantly in the pigmented form of urticaria, but there is one form, characterized by a series of dark-yellowish or purplish-red macules which are somewhat itchy, in which no mast-cells are found. When these macules are irritated by scratching there occurs an elevation of the skin which very closely resembles urticaria.

The diagnosis of urticaria is usually not difficult. The important features are the special burning, itching sensations that accompany the occurrence of raised patches which have what are called geographical boundaries,—that is, sharply marked edges. The fact that the disease occurs over limited areas of the body and is non-symmetrical, and the suddenness with which it develops, are characteristic signs; its relatively rapid disappearance and its frequent relapses make the diagnosis certain. The diagnosis of urticaria of the tongue or palate must be made sure by the observation of cutaneous patches of œdema, or by the knowledge of some special cause for the disease.

Pigmented urticaria may easily fail to be recognized if seen in the intervals between the occurrence of the elevated lesions characteristic of true urticaria. At times these can be produced by irritation of the pigmented area, but this is not always the case. If there is serious doubt, and a diagnosis is absolutely necessary for some reason, a small portion of the tissue may be removed and examined for the so-called mast-cells. Hemorrhagic urticaria is not easy to distinguish from hemorrhagic erythema, but the two affections are really only different forms of very similar pathological processes in the skin.

The prognosis of urticaria as regards life is not serious, if we except the cases in which urticaria is said to be the causative factor in the production of fatal œdema of the glottis. Yet the disease may prove extremely annoying, and may cause the patient much misery for a long time. The greatest discomfort is likely to be caused by the forms of the disease due to the taking of some sort of food for which the patient has an urticarial idiosyncrasy; the cleansing of the digestive tract by vomiting and purging will usually relieve this without delay. With regard to the prognosis as to

relapses little can be said. There are cases that continue to have recurring attacks for many years, notwithstanding the greatest care in diet and habits of life; but as a rule proper treatment of the patient will stop the acuter forms of the disease in a few weeks at most. The pigmented forms are almost hopelessly obstinate to any ordinary treatment, and successive attacks may make the pigmentation more and more of a serious disfigurement. Very little can be done for the chronic persistent form of which I have spoken, which may cause very serious deformities and make the patient a really pitiable object to see, though there is but slight discomfort beyond the annoying presence of the deformity.

The treatment of urticaria must be directed to meet the two main indications,—the discomfort and the underlying cause of the disease. French authorities have shown that the use of bandages is the best means to prevent the development of the urticarial elevations, and also to combat the pain and itching which they cause. These bandages should be applied over bits of cotton, which are kept in place by the winding of the bandage. There is one objection to this form of treatment,—it cannot be employed to any extent, at least it cannot be maintained for any length of time, especially during warm weather, and urticaria is quite as liable to occur in summer as in winter. Consequently it is only when the pain and itching are a veritable torment that bandaging should be employed.

A host of local applications have been suggested for urticaria. Those which have given the best results are made with a basis of balsam, to which has been added about one-thirtieth its quantity of menthol and about one-tenth of beta-naphtol. Vaseline with ten per cent. of naphtol has produced a very good effect in relieving the discomfort and the itching. Salicylate of methyl, especially in the form of oil of wintergreen, has also been of distinct service in this respect. Washes made of acetic acid to which a little chloral or ether is added, are useful, as are also dilute solutions of carbolic acid. The preparation of carbolic acid known as steresol has been highly recommended of late. Compresses dipped in a five per cent. solution of salicylate of sodium have been known to give good results. Oxide of zinc in the form of paste may be employed, and when put on as directed by Unna may be allowed to remain for a considerable time, producing a more or less continuous effect. Varnishes made with ichthyol—especially such as have a tendency to

contract on drying, so that they exert the mechanical effect of pressure, as well as a remedial effect due to the medicaments they contain—have been found very satisfactory by some.

Whenever urticaria occurs because of the presence of irritating material in the intestines, or because certain food materials have been ingested which are known to occasion the disease either in the particular individual or in general, a purgative should be employed. Even when it is not known for certain that any such irritating material is in the digestive tract, the use of intestinal antiseptics on general principles is indicated. For this the salicylate of sodium, in doses of fifteen grains three or four times a day, has been especially recommended.

When dyspepsia or digestive disturbances of any kind are present they must be cured or decidedly improved before we may confidently expect a cure of the attacks of urticaria or of the tendencies to the affection between the attacks. For the relief of urticaria caused by dyspepsia due to fermentation the rational measures are, careful regulation of the diet and the internal use of iodides, of sulphur or of fluoride of ammonium, or of beta-naphtol or some other intestinal antiseptic that has been known to give good results. Even when there are no signs of dyspepsia present it is well to suspect a dyspeptic origin for the affection and to treat the intestinal tract on this principle.

Unna has obtained some good results in the treatment of urticaria by doses of atropine up to its physiological effect, with the idea that it will relieve the angioneurosis which in his opinion is the proximate cause of the attack.

It must be understood, of course, that in every case of urticaria every kind of food and all medicinal substances which can by any possibility provoke an attack of the disease should be avoided very carefully, and it is well to remember how varied these substances are. Not infrequently it will be found that despite the fact that the patient himself does not suspect it, he is taking some substance which is responsible for the development of the lesion. As soon as this is discovered it must be eliminated from his dietary.

TREATMENT OF DEAFNESS BY DIRECT MASSAGE OF THE OSSICLES OF THE EAR.

A SPECIAL ARTICLE.

BY DR. CHARLES J. KOENIG,

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THERE are a certain number of rather serious and discomforting ear symptoms for the relief of which all the ordinary methods of treatment known at the present time have been unable to accomplish very much. The most prominent of these are tinnitus aurium, the subjective hearing of noises of various kinds in the ear, and deafness, or the failure to hear external noises, either completely or partly. It is now recognized that these symptoms are almost hopelessly persistent in many cases and any form of treatment that holds out a reasonable promise of relief is therefore especially welcome.

Both of these symptoms, and particularly the existence of partial deafness, are in many cases due to the presence of inflammatory products in the delicate tissues that make up the conducting apparatus of the middle ear. Even slight chronic inflammation in the joints connecting the minute ossicles of the ear may so impair the conduction of external impressions as to cause deafness. The subjective noises that are heard in the ear, and of which patients often complain bitterly, are not infrequently due to the same cause.

Various methods of massage for the purpose of rendering the joints connecting the ossicles of the ear more easily movable have been suggested from time to time. Some of them have given more or less satisfaction, and others have failed to accomplish the results announced except in their inventor's hands. Lucae's invention of a spring sound, used in the ear in order to produce direct massage of the ossicles, has quite recently given excellent results in a number of reported cases. Of this instrument I shall have occasion to speak, and I desire to suggest certain improve-

ments in the technic of its employment that will render its use less painful to the patient and more satisfactory to the physician.

There are a number of conditions in which the use of this spring sound is indicated. Among these may be mentioned the following:

1. Ankylosis of the joints of the ossicles, either complete or partial, due to an incomplete adhesive inflammatory process in the minute joints between the little bones.

2. The presence of cicatricial tissue, resulting from acute inflammation of the middle ear and hampering the action of the conducting apparatus by firmly binding it together, or by rendering the movements of the separate parts more limited than normal or perhaps impossible.

3. Fibrous sclerosis of the middle ear. By this is meant not bony sclerosis, but the presence of fibrous tissue due to a chronic inflammatory process.

4. Nervous deafness, especially the functional deafness that occurs without any known or recognizable pathological basis. It is intermittent in character, coming and going without very evident reasons. It occurs particularly in hysterical patients and is met with in other forms of functional nervous diseases.

5. Tinnitus aurium. Not all cases of subjective sounds in the ear are relieved by massage of the ossicles. Those, however, which are dependent upon nervous conditions and do not involve serious pathological lesions of the vascular supply of the ear or of the nervous system, peripheral or central, are almost invariably benefited by the use of Lucae's sound.

It is probable that the benefits resulting from the properly regulated employment of Lucae's instrument come not only because of the massage of the ossicles, but also because it sets up a series of oscillations in the endolymph and perilymph of the circular canal. These oscillations effect a stimulant massage of the terminal nerve-endings of the acoustic nerve, and thus render them more sensitive and more capable of conveying sensation than they were before. It is not improbable, too, that the movements produced in this fluid serve to modify the nutrition of the terminal nerve-endings and so render them more capable of fulfilling their function.

Lucae's spring sound (Fig. 1) consists of a small cylindrical

barrel, containing a spring, to which a steel sound, hollowed at the free end, is fixed. Into this hollow end is inserted a small cup made to fit the processus brevis of the little ear bone called the malleus, or hammer. The spring in the cylinder handle serves to make the process of massage very gentle, and enables the operator to act upon the processus brevis without undue pressure. The presence of the spring allows the exertion of only a limited amount of force. There is a cap on the distal end of the handle which can be screwed from left to right and which gradually increases the pressure that may be exerted by the instrument from sixty to one hundred grammes. This is an extremely ingenious instrument, and with the suggested modification its application will become greater than before, and its power for good will be so much enhanced as to make familiarity with it of particular service to practical specialists in diseases of the ear.

Fig. 1.

Lucae's Spring Sound.—1, small cup to fit processus brevis of malleus; 2, steel sound running into the cylinder, surmounted by a small cup that is dipped into paraffin in the new technic; 3, cylinder containing the spring on which the steel sound is fixed; 4, cap that screws from left to right in order to increase the pressure from sixty to one hundred grammes.

How long the sound should be employed and the length of each séance are questions which must depend upon conditions existing in each individual patient. It may require treatment every second or third day, and lasting for several months, to produce any noticeable improvement in the hearing of patients suffering from sclerosis of the middle ear. In these cases the ankylotic conditions must be loosened up, the contractile tissue rendered more elastic, and the conduction generally restored. As a rule, the pathological condition that constitutes the main reason for the deafness in these cases has existed for a long time. Immediate relief, therefore, cannot be looked for, and the patients themselves will readily realize that improvement, to be lasting, must be effected by slow degrees.

On the other hand, in certain nervous cases, especially in those forms of deafness which develop as the result of hysterical manifestations, one sitting may prove effective in enabling the patient

to hear sounds that were previously absolutely imperceptible. This is also true of tinnitus aurium due to nervous causes or developing in a nervous patient. I have more than once seen such cases so much improved by one application of the spring sound that a very favorable prognosis was possible at the end of the first sitting.

This form of treatment should be tested in all cases of sclerosis of the middle ear before submitting them to any serious operative interference. The method employed is quite simple; it gives rise to some discomfort, but it can do no harm in any case. In mild cases, at least, its benefits will be soon observed, and the patient will be thereby encouraged to proceed further with it. Other methods of treatment need not be neglected while the spring sound is being employed. The use of electricity, for instance, in conjunction with the direct massage of the ossicles, may be very beneficial; iodide of potassium taken regularly for a long period of time may also prove of service. It is needless to say that the condition of the nasopharynx must not be neglected. It should be carefully and frequently cleansed, and any obstructions to respiration or to the functions of the Eustachian tube that may exist should as far as possible be removed. It is only by putting the patient in the most favorable condition for the exercise of hearing that benefit from the employment of the spring sound can be expected.

The chief disadvantage in connection with Lucae's spring sound is the fact that, in the great majority of cases, considerable discomfort accompanies its use. This is the experience of practically every person who uses the instrument; and the results are the same even when it is applied with extreme delicacy of touch. This effect is to be expected, for the contact of a metallic instrument with the processus brevis of the hammer is sure to cause pain in ordinary cases. Occasionally, it is true, patients are found whose exceptionally insensitive organizations are not affected by the employment of the sound; but these are rare and isolated cases. It is for the ordinary class of patients that our therapeutic measures must be arranged.

If local anæsthesia of the membrana tympani could be obtained by means of solutions of cocaine, or if the mixture of cocaine, carbolic acid, and menthol recommended by Bonain for use as a local anæsthetic could be successfully employed in this connection,

the pain caused by the application of the sound would be prevented. Even in this event it is probable that some of the discomfort of the direct massage would still remain. We have reason to believe that the passive movement of the ossicles and of their joints, even when accomplished with great delicacy, may be a source of considerable pain, especially when the pathological condition that underlies their immobilization has existed for some time. Local anæsthesia of the membrane of the ear-drum by applications of cocaine to its external surface cannot be expected, in the majority of cases, to extend through the drum-head so as to include in the area of anæsthesia the ossicles and their joints.

Otologists are familiar with the fact that applications of cocaine to the membrana tympani for the purpose of producing complete anæsthesia are very unsatisfactory. This has been our experience lately while testing the application of the various methods suggested for massage of the drum-heads. Personally I have never succeeded in obtaining anæsthesia of the membrana tympani any more effectually than the local anæsthetic effect of cocaine can be obtained by applying the drug to a part of the cutaneous surface not deprived of its epithelial covering.

I have not been any more fortunate in securing anæsthesia of the drum-head with the mixture of Bonain than with that of Grey. Aniline oil enters into the composition of the latter mixture, which is supposed to have the property of rendering pavement epithelium permeable for other medicinal substances, and especially the anæsthetics with which it may be associated. It has not in my hands fulfilled the expectations founded on this theory. I have even at times feared that the aniline oil might have a local toxic effect, and thus increase the necrosing action which cocaine produces on delicate superficial epithelium.

I think that those who claim to have obtained satisfactory local anæsthesia by these various applications must have exercised at the same time considerable persuasive ability, and that they should perhaps acknowledge the influence of suggestion in obtaining the anæsthetic effects quite as much as that of the local anæsthetic employed. Patients who are sensitive to suggestion are, unfortunately, in the great minority. Though it has been put forward more than once, we cannot raise suggestion to the dignity of a method of anæsthesia for current use, and we are therefore obliged

to find something more definite and more likely to produce the desired effects in the ordinary circumstances of practice.

Besides the pain, Lucae's sound is apt to produce small excoriations of the epithelium. It is not impossible that these small denudations of the epithelial surface of the drum-head should lead to infection, and as a consequence myringitis might develop, or even a deeper and more dangerous inflammation of the structures of the middle ear, with all the evil consequences which such a condition might imply. In one case, as the result of a careless use of the sound, I observed a myringitis, and in another the production of a small pink-covered bulla, containing a sero-sanguineous fluid.

Because of these various inconveniences I should have abandoned the use of Lucae's sound entirely—though, I may add, with considerable regret—had I not found an easy, simple, and practical means of rendering its use tolerable, and at the same time overcoming its other disadvantages, by the employment of the following technic: I melt a small portion of paraffin in a minute crucible, and into this I dip the cup attached to the upper end of the sound and which is so constructed as to receive and cover the processus brevis of the hammer. In a few seconds after removing it the small portion of paraffin which remains adherent to the cup cools and solidifies. If this be now pressed against the processus brevis, the latter will sink deeply into the paraffin, which must be sufficiently soft to be moulded to the proper shape. The contact between the cup and the processus brevis is very close, and there is consequently during its use no grazing over the part of the drum which covers this portion of the hammer. Care must be taken that the paraffin shall not become too hard, for in that event the desired object would be in part defeated. Experience will soon teach one to recognize the degree of consistency especially suitable for the purpose. The paraffin does not irritate, and therefore massage becomes almost painless. As it does not produce the slightest excoriation or even frictional irritation of the drum membrane, the massage can be prolonged, and is consequently more efficacious.

Patients on whom I previously used the sound without paraffin and afterwards with it have been astonished at the difference produced by this simple expedient. While they, as a rule, dreaded very much the use of the sound before the introduction of the paraffin modifications, they now declare that the massage is rather

easily borne and produces no serious discomfort. Of course, in the first sitting, even with the paraffined sound, the same care and prudence must be exercised as with the non-paraffined sound,—that is to say, the operator should make only a few forward and backward movements and should begin with a pressure of sixty grammes. If some discomfort is felt and if the ear afterwards feels sensitive and sore, the massage should not be repeated for a day or two. After awhile it may be repeated every day. If circumspection and tact be used, the drum and the ossicles will become quite accustomed to the contact of the instrument, and then the number of movements and the pressure can be gradually increased. As I have already said, by screwing the small cap at the manual end of the sound from left to right, the pressure exerted by the instrument can be gradually increased from sixty to one hundred grammes.

I believe that great good can be accomplished by the use of the spring sound thus modified. It is especially useful in such cases as are ordinarily almost utterly obstinate to treatment. In ankylosis of the ossicles there is a distinct indication to be met by the use of the sound, and its employment is a precious part of rational therapeutics. I may repeat once more that no harm can come from its employment, while in a number of cases almost marvellous results have been obtained from its careful application.

PRACTICAL HINTS.

WATER IN PNEUMONIA.

Some years ago the drinking of water was considered by most medical men as unsuitable for those suffering from pneumonia. Its abundant use is now, however, very generally insisted upon from the very beginning of the attack, the temperature of the water being regulated to suit the patient. Water is the best diuretic that we have, and its employment prevents the constitutional depression incident to the absorption of the toxins from the pneumonic process in the lungs, and favors free intestinal evacuation. As soon as fever causes the patient to become uncomfortable, he should be sponged with cold water. Some patients become restless under a temperature of 101½° F., while others are quite comfortable at 102½° F. If the signs of toxæmia begin early in the case, venesection, followed by the introduction of a corresponding amount of salt solution, is the most helpful method of treatment, and is, in fact, only another form of hydrotherapy.

THE FOURTH DISEASE.

The most interesting diagnostic problem at present occupying the attention of medical men is the question of the existence of a fourth disease in the measles-scarlet-fever group. Even those conservative diagnosticians, who are unwilling to admit the existence of a fourth disease, concede that there are two extreme variations from the average type of German measles or rubella. For these two variations, the names rubella scarlatiniformis and rubella morbiliformis have been suggested. Positive evidence as to either of these forms of German measles proving protective against the other form is too scanty to support any conclusion as to the diseases being identical or independent. There is much more involved in this question of the admission of a fourth disease in this group of exanthemata than is usually thought. The number of our exanthematous diseases has been increasing steadily since the beginning of the last century. It must be remembered that a hundred years ago even measles and smallpox were not yet well differentiated from

each other, and as for scarlet fever and diphtheria, they were, as yet, entirely undifferentiated. There has been, in many places in this country, a tendency to insist that there may be a third disease in the smallpox group, occupying a middle position between chickenpox and true smallpox. There are eleven distinct types of nutgall eruptions produced upon leaves by the stings of various insects. Each eruption is distinctive for the special insect that makes it, though, in its general character, it is similar to others. Cutaneous eruptions of the exanthemata in the more complex human skin may well prove of interest for clinical investigators.

CONJUNCTIVAL ASTHMA.

Rosenbach has recently described a form of hyperæmia of the conjunctiva which occurs especially in neurotic patients, or, at least, in those suffering from severe nervous symptoms at the time of its development. An intense hyperæmia, particularly of the upper lids, comes on at intervals, and is usually accompanied by a hyperæmia of the mucous membrane of the nose. Patients complain of a sense of extreme weariness and a heaviness of the head; very often they are restless during the day and unable to sleep at night. Usually they are very much depressed and inclined to be melancholic. A sense of constriction about the neck, somewhat similar to the sensation which comes from wearing a tight collar, and a feeling of oppression of the chest, with a tendency to frequent sighing, are also noted. A weak solution of cocaine gives some relief, lasting only as long as the local action of the cocaine can be noted. The ordinary local remedies for conjunctivitis, boracic acid, or weak solutions of nitrate of silver, or other means, are completely ineffective. Rosenbach thinks that the condition is associated with cerebral hyperæmia which causes the severer nervous symptoms. Intestinal disturbances, especially hemorrhoids, would seem to be etiologically connected with the disturbance of the circulation. All of the five patients under observation were rather stout, and at least three of them were distinctly plethoric. They were between forty and fifty years of age, and in the women, the nervous manifestations of the menopause were evidently part of the symptom-complex of the affection. Now that true essential asthma has come to be looked upon as a neurosis, or an angioneurotic œdema of the lungs, the description of other localized condi-

tions of œdema, due to neuroses of the blood-vessels, is interestingly suggestive.

SURGICAL RELIEF FOR BRONCHIECTASIS.

Surgical intervention for pulmonary disease is becoming much more active. Putnam recently presented a case at a meeting of the Harvard Medical Society of New York City, in which he had evacuated several bronchiectatic cavities that gave out profuse and extremely malodorous secretions. Indeed, the odor was so foul that even the little patient's mother could scarcely bear to sleep with her child. Although more than a year has passed since the operation, there has been no recurrence of this extremely annoying symptom, notwithstanding the existence of other dilatations of the bronchi. The cavities were evacuated by means of the Paquelin cautery at a low, red heat. This required the operation to proceed slowly and necessitated the frequent withdrawal of the cautery in order to burn it clean by raising it to a white heat. The situation of the cavities containing the fetid secretion was exactly located by means of an aspirating needle, which was then allowed to remain *in situ* to act as a guide for the subsequent operative procedures. After the completion of the operation, the intrathoracic wound healed very satisfactorily, and there were no complications in the lungs themselves, notwithstanding the fact that through a mistake water for irrigation purposes was allowed by an assistant to flow into the wounded pulmonary tissues and appeared at the mouth.

CAUTERIZATION BY HOT AIR.

In the interesting article written by Holländer and published in Volume II., Ninth Series, of the INTERNATIONAL CLINICS, are to be seen pictures of certain cases of lupus that had been cured by being subjected to very hot air. Schwabe uses a form of apparatus which resembles a Paquelin cautery. He considers that for cauterizing purposes hot air is much more under the control of the operator than is heated metal of any kind, and that it produces much less pain. When the stream of hot air first touches the skin the latter becomes white, changing after a moment to light gray. If the hot air is allowed to act too long, the tissues begin to steam and char. The light gray stage is sufficient for all therapeutic purposes of cauterization. The subsequent sore is only small in amount, does not take long to heal, and leaves no scar.

IPECACUANHA SPRAY FOR WINTER COUGH.

Ringer, the English authority in therapeutics, who has been the originator of many practical suggestions for the treatment of obstinate affections has recently recommended ipecacuanha spray as an efficient remedy for the persistent coughs of chronic bronchitis in the winter time. The remedy is employed in the form of wine of ipecacuanha in an ordinary handball spray apparatus or atomizer, either pure or diluted with two or three times the amount of water. During the spraying the patient is directed to inspire deeply and the nose is closed with the fingers. At the beginning a drachm of the drug is employed for one application; later this amount may be increased to three or four drachms. The patient is directed not to swallow any of the spray which accumulates in his mouth. This method of treatment is said to relieve the dyspnœa and feeling of tightness across the chest better than any other. It loosens the secretion, encourages expectoration, makes the patient much more restful, and seems to shorten the duration of the bronchitis. At first the remedy must be used with care, because it may cause vomiting. The treatment is not recommended in true essential asthma, but on the contrary is contraindicated in this affection because it causes aggravation of the symptoms.

RENAL PERCUSSION.

Goldflam recommends a new method of examining the kidney which has now been used with satisfaction by a number of physicians in many parts of the world. The patient is seated or stands with his back to the examiner in a slightly stooping posture. The doctor strikes lightly with the ulnar border of the fist on the lumbar region, either perpendicularly to the mass of the lumbar muscles, or slightly obliquely. The blows are repeated so as to cause a series of shocks that are not painful to healthy individuals, but are painful in such renal diseases as stone in the kidney, pyelonephritis, nephritic neoplasms, and tuberculosis of the kidney. Of course, in certain very sensitive individuals even slight blows may be complained of so much as to make the diagnostic significance of this method of examination fallacious. As a rule, however, this percussion enables the examiner to differentiate kidney troubles of various kinds from such other affections as biliary colic, or tumor of the spleen, or of the stomach, for these organs are never painfully affected by the series of shocks over the sacrolumbar region.

ABORTIVE TREATMENT OF STYE.

Where a number of styes (hordeola) occur there is sure to be some factor at work lowering the vital resistance of the ocular tissues. Usually styes are due to eye-strain, and there is present a refractive error that requires correction before the tendency to styes will be overcome. At times, patients will be found to be doing their work under conditions of illumination that are unsuitable, either the light is too glaring and shines too directly into the eyes or the illumination is insufficient for the work they are trying to do. When arc-lights were first introduced, they were especially unsteady, and it was noted that many persons who worked under them suffered from styes. As a rule, patients who get many styes will also be found to be suffering from a condition of anæmia, or from some blood dyscrasia that leaves their tissues especially liable to invasion by the ordinary pus cocci. It must not be forgotten that a stye is, after all, a sort of boil on the margin of the lid. When anæmic conditions are present, the bowels should be kept freely opened and the internal use of the iodide of iron should be employed. For the abortion of a stye that is just beginning, the removal of the eyelash which occurs as a rule in the centre of the inflammatory process is the initial step. If no pus has formed as yet the swelling should be touched with tincture of iodine, or with pure carbolic acid. If this is applied carefully there need be no fear of any injury or even the slightest irritation to the conjunctiva. Warm applications usually serve to lessen the pain and to hasten the progress of the stye. Once pus has formed it should be evacuated by a small incision. The inflammatory thickening which remains and which is not only unsightly, but a persistent source of irritation for some time, may be encouraged to resolve by applications of a weak yellow mercurial ointment. Casey A. Wood insists on the frequency with which styes occur in anæmia and especially in chlorotic girls. He recommends a carefully selected diet, plenty of fresh air, and a tonic containing the tincture of the chloride of iron, phosphoric acid, tincture of rhubarb, quinine, and strychnine.

PROSTATIC INFLAMMATION AND HYPERTROPHY.

The enlargement of the prostate seen so often in elderly men is beginning to be looked upon as nearly always due to chronic

inflammatory conditions of the prostate earlier in life. There are, it is true, genuine glandular enlargements partaking of the nature of adenomata, which are not of this class. Most of the hypertrophies of the prostate, however, that are represented by a large increase in the connective tissue of the organ and by a correspondingly enlarged and indurated prostate, are now looked upon as due almost as a rule to chronic urethritis. The best prophylaxis against the development of enlarged prostate late in life is, therefore, the careful treatment of acute urethritis, and especially of the chronic urethritis that is so often seen. This does not necessarily mean meddlesome treatment of the urethritis, and especially does not involve the use of frequent injections or of large irrigations given at pretty high pressure. Meddlesome treatment is probably accountable for many of the prostatic complications and gives rise to the subacute and chronic inflammatory conditions which lead to subsequent enlargement of the prostate.

PROSTATIC ABSCESS.

The impression is gaining ground that suppuration of the prostate is more frequent than practitioners have believed. Heretofore, it has been the custom to wait for the development of enough pus in the prostate to enable the palpating finger in the rectum to obtain fluctuation before an operation for the condition was considered advisable. Alexander, of New York, points out that if this symptom is waited for the pus is very liable to find its way outside of the prostate and thus cause extensive suppuration in the perineum and even in the ischiorectal fossa. He suggests the attempted evacuation of the pus when there is marked albuminuria without any kidney lesion to account for it, when one lateral half of the prostate is notably larger than the other, and when there are some urinary symptoms with pain and a febrile condition besides tenderness of the prostate. He does not advise the use of the incision just in front of the rectum, but opens the urethra through the perineum and then evacuates the pus through the opening thus made, the finger inserted in the urethra serving as a guide for the location of the prostate.

Medicine

A CASE OF SPLENOMYELOGENIC LEUCOCYTHÆMIA SUBSEQUENT TO AN ATTACK OF MALARIAL FEVER.

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Although the title of this paper is "A Case of Splenomyelogenic Leucocythæmia subsequent to an Attack of Malaria," I will ask your indulgence for a short time while making a few preliminary remarks.

Leucocythæmia, or leukæmia, has been known to medical science for the past fifty or sixty years, having been first observed during life by Dr. Fuller in 1846. To Dr. Hughes Bennett, who in 1845 published a case diagnosed after death, we owe the name "Leucocythæmia," while Virchow, who in the same year published a case more carefully worked out than that of Dr. Bennett, calls the malady "leukæmia," or "white blood," a designation obviously not strictly accurate.

The definition of the term leucocythæmia has undergone several changes within recent years. For example, in Quain's "Dictionary of Medicine," second edition, 1895, it is defined as "a chronic disease, in which there is a considerable and permanent increase in the number of the pale blood-corpuscles; usually associated with enlargement of the spleen, sometimes also with that of the lymphatic glands, and with disease of the medulla of bone." Recent observations tend to show that some cases of leucocythæmia run an acute course, terminating fatally in a few weeks (the blood of such a case is shown in Plate II.); hence it can scarcely be defined as a "chronic disease." Then, secondly, cases of leucocythæmia are on record in which during some part of their course the *number* of the leucocytes was little, if at all, increased, but in which the *proportion* of the various white cells in the blood is different

from that normally present; in other words, the change is *qualitative,* but may not always be *quantitative.* Bearing in mind these facts, leucocythæmia may be defined as "a disease, generally chronic, occasionally acute, in which there is a marked *qualitative* alteration in the white blood-corpuscles, generally associated with a considerable and permanent increase in their number; usually, too, associated with enlargement of the spleen and of the lymphatic glands and with disease of the bone marrow."

Leucocythæmia is not a very common disease, neither is it one of the *raræ aves* of medicine, as, for example, acute yellow atrophy of the liver. On an average two or three cases of the disease are seen in University College Hospital every year; thus, during the years 1896 to 1900 eleven cases of leucocythæmia have been in the hospital, two of which were of the acute lymphatic variety.

Varieties of the Disease.—Two forms of the disease are now generally recognized,—(1) the splenomyelogenic and (2) the lymphatic,—sometimes known as the *myelocytic* and *lymphocytic* forms respectively. The names "splenomyelogenic" and "lymphatic" have reference to the organs or tissues which are possibly at fault, —namely, the spleen and bone marrow in the one case, and the lymphatic system or lymphoid tissue in the other. In the present incomplete state of our knowledge as to the causation and pathology of the disease, these names are open to objection, and it is preferable to use the terms "myelocytic leucocythæmia" and "lymphocytic leucocythæmia," as indicating which variety of white corpuscle is present in the blood in relatively greater numbers. There is, however, an objection to the use of these terms, for in both forms of the disease the two varieties of leucocyte may be relatively as well as absolutely increased, and indeed some cases present the clinical aspects as well as the blood condition of both forms,—the so-called "mixed cases." Some cases terminate fatally in a few weeks, while others go on for two, three, or four years after the patient first seeks advice, and in all probability have really a far longer duration. The former type of case is the acute, the latter the ordinary or chronic form of the malady, and it is possible to have acute and chronic examples of both the lymphatic and the myelogenic varieties. The very acute cases, fatal in from two to six or eight weeks, have, I think, all been of the lymphatic type, and have only recently been recognized as being cases of leucocythæmia.

A very interesting series of five cases of acute lymphatic leucocythæmia, all of which were in University College Hospital, has been collected and carefully described by Drs. J. R. Bradford and H. Batty Shaw, in the Medico-Chirurgical Transactions" (1898, vol. lxxxi). As the condition is an interesting one and liable to be overlooked, I should like to give you a brief sketch of the clinical aspect of the cases, although at present we can, unfortunately, not avert a fatal issue, even though we make a correct diagnosis. In many of the recorded cases the symptom for which the patient seeks advice is a stomatitis, often very severe, and which may lead to cancrum oris and necrosis of the jaw. With this are associated general symptoms, such as malaise, headache, and advancing anæmia; in most cases, too, the superficial lymphatic glands are enlarged, especially the cervical, axillary, and inguinal. The spleen was usually found enlarged and the bone marrow diseased in most cases. The blood is very characteristic: there is a considerable increase of white corpuscles, consisting mainly of large lymphocytes or mononuclear leucocytes. These may constitute ninety per cent. or more of the total number of white corpuscles, the other varieties being not only relatively but actually decreased. Plate II., Fig. 2, is an actual field from a blood film of a child who died of lymphatic leucocythæmia. The number of white corpuscles present was one hundred and thirteen thousand per cubic millimetre, of which 99.2 per cent. were mainly *small* lymphocytes and only 0.48 per cent. polymorphonuclear leucocytes. Two interesting features in the case were the short history—the child is said to have looked quite well two or three weeks before death—and the fact that *post mortem* the kidneys were found much enlarged, owing to diffuse lymphocytic infiltration, especially of the cortex.

Causation.—When we come to inquire into the causation of the disease, it is advisable to consider the two forms of it separately, and a more intimate knowledge of the subject may show eventually that the disease now known as " acute lymphatic leucocythæmia" is of an entirely different nature from the ordinary type,—the chronic " splenomyelogenic" leucocythæmia. Ehrlich says, " Acute lymphatic leukæmia, characterized by its rapid course, small splenic tumor, the tendency to petechiæ and to the general hemorrhagic diathesis, has given all observers the impression of an acute infectious process." It is very probable that, as in the case of most

infectious diseases, a micro-organism will be found to be the cause of the disease, but I am not aware that any specific germ has hitherto been found in the blood or tissues of these patients. The second form of lymphatic leukæmia is marked off from the preceding by its chronic and often very protracted course (Ehrlich). The blood condition may resemble that found in the acute cases and the spleen shows its participation in the disease, as a rule, by very considerable enlargement. Ehrlich assumes with regard to the origin of lymphatic leukæmia that "the increase of the lymph-cells is brought about by a passive inflow into the blood and not by an active emigration from chemical stimuli," for experimental research has shown that chemotactic reaction produces a leucocytosis of the polymorphonuclear elements alone. There is no doubt that the lymphocytes found in the blood, both in health and in disease, arise from the lymphatic system and are identical with those of the lymphatic glands and the rest of the lymphatic system.

There is indeed a very close resemblance between the lymphocytes found in the blood in lymphatic leucocythæmia and the cells seen in some forms of sarcomata. This has led to the theory that lymphatic leucocythæmia is a "sarcoma of the blood," the only difference being that in ordinary sarcoma the cells are stationary within the tumor, whereas in leucocythæmia the "sarcoma" cells are circulating in the blood,—the tissue affected.

Sir Samuel Wilks has called the disease *arterial pyæmia,* regarding the leucocytes in the blood as ordinary pus-cells. This view, however, cannot be maintained, for in pus the large majority of the leucocytes are of the polymorphonuclear variety, whereas in leucocythæmia the other varieties of white cells are in the majority, and the ordinary polymorphonuclear cells are relatively greatly diminished, and may even be present in absolutely smaller numbers than in health.

Myelogenic leucocythæmia presents a different clinical picture, and the blood is very characteristic, owing to the pressure of large numbers of large mononucleated granular cells known as *myelocytes.* These cells are absent from healthy blood; they occur normally only in the bone barrow and, according to Ehrlich, have nothing whatever to do with the spleen or lymphatic glands. The myelocytes are regarded by this observer as the early stages of the ordinary polymorphonuclear leucocytes, and he explains the origin

of leucocythæmic blood "by the emigration into the blood, *under the influence of the specific leukæmic agent,*[1] not only of the formed polynuclear elements, but also of their mononuclear early stages (myelocytes), and so classifies myelogenic leucocythæmia with the active leucocytoses." In this explanation of Ehrlich the words "under the influence of the specific leukæmic agent" naturally lead to the question as to what the agent may be. This question cannot be answered with any certainty at present. In some cases an injury or blow over the spleen seems to have been the exciting cause, in others worry, and in others again a strain. By some observers it is thought to be a specific infectious disease, but different organisms—cocci and bacilli—have been described by different investigators. Löwit has described forms like plasmodia in the white corpuscles in myelogenic leucocythæmia, but failed to find them in the lymphatic variety. Hitherto no one has been able to reproduce the disease, nor have inoculations of leucocythæmic blood into animals given any consistent results. Sir William Gowers many years ago carefully investigated the antecedent history in one hundred and fifty cases of leucocythæmia, and found that in thirty of them the patients had previously suffered from some form of malarial fever, from a few months to twenty-five or even thirty years before the recognition of the leucocythæmic condition; in nine others there was no history of any malarial attack, but the patients had lived in a malarious district; thus, out of one hundred and fifty cases, thirty-nine, or more than a quarter of them, gave a history of antecedent malaria or exposure to its influence. Many subsequent observers have failed to find an antecedent malarial history in such a large percentage of the cases, and of the eleven cases which have been in University College Hospital during the past five years, in one only (nine per cent.) is a malarial history given. Then, again, it appears to me that in these malarial cases some other factor must operate, for I have seen several enlarged malarial spleens and heard of many more cases of patients who have been in the tropics and had malaria, whose blood nevertheless has shown no increase of white cells and who have had no signs of leucocythæmia.

[1] The italics are not in the original; "Histology of the Blood," by Ehrlich and Lazarus, translated by W. Myers, Cambridge, England, 1900.

The Blood in Leucocythæmia.—I do not here propose to go into the clinical aspects of leucocythæmia, but will confine my remarks to the blood changes seen therein, and will just mention one or two symptoms of the disease occasionally met with and not usually given in the text-books. The first symptoms may be those of renal colic, due to the irritation of the renal tubules by the excess of uric acid frequently passed by patients with leucocythæmia. Sometimes a patient with the disease comes to the physician complaining of asthmatic attacks, and women may come saying they are pregnant.

Before describing the blood changes seen in the disease a few remarks about the leucocytes normally found in the blood may not be out of place. The number of white corpuscles in the blood varies considerably under physiological conditions: it may be anything between four thousand and ten thousand per cubic millimetre. In children it may be even higher in apparently healthy conditions. The digestion of a meal causing a notable physiological increase in the number of leucocytes, it is important always to examine the blood as long as possible after the last meal, as, for example, before breakfast or at about twelve o'clock midday.

The varieties of white cells found in blood are as follows:

1. Small lymphocyte (Plates I., II., *g*). Cells about the size of a red corpuscle, with a large, usually round, nucleus almost filling the cell, little protoplasm containing no granules. Normally this variety forms from twenty to thirty per cent of the total number of white corpuscles.

2. Large lymphocytes or large mononuclear leucocytes (Ehrlich) (Plate II., *k*). Cells two or nearly three times as large as the red corpuscles, with a large nucleus, frequently oval and excentrically situated, relatively smaller than the nucleus of the small lymphocyte. The nucleus usually stains feebly, and the protoplasm, which is relatively large in amount (compared with the small lymphocyte), usually stains more feebly than the nucleus. No granules are present in the protoplasm. Ehrlich says that they form only one per cent. of the total number of leucocytes in normal blood, and that they are not derived from the "small lymphocytes," but that they change in the blood to "transitional forms" with indented or oval nucleus and scanty neutrophilic granulations. The two varieties together (large mononuclear leucocytes and "tran-

sitional" forms) constitute from two to four per cent. of the white corpuscles, according to Ehrlich. Cabot groups them together as "large lymphocytes" and says they form from four to eight per cent. of the total number of leucocytes in normal adult blood. In infants the leucocytes (large and small) are more numerous than in adults and may constitute from forty to sixty per cent. of the total number of white corpuscles.

3. Polymorphonuclear leucocytes ("polynuclear neutrophiles" of Ehrlich) (Plates I., II., *a*). Cells about twice as large as the red corpuscles (diameter from twelve to fifteen microns), with a very irregular contorted nucleus, often resembling the letters S, Y, E, or Z in shape. The nucleus often appears multiple, but on careful focussing one can usually make out fine connecting bands between the different parts of the nucleus. The nucleus stains deeply with hæmatoxylin, methylene blue, methyl green, and similar dyes. The cell is filled with fine, irregularly shaped granules, oxyphile (or, according to Ehrlich, neutrophile) in character. The number found in normal adult blood is from sixty-two to seventy-two per cent. of the total number of leucocytes. In infants the percentage is smaller and may be between twenty and forty per cent. of the total leucocytic count. These cells are in the main produced fully formed in the bone marrow and emigrate thence to the blood, and, according to Ehrlich, they are also derived in small part from the "transitional cells" within the blood stream. This variety of leucocyte is actively amœboid and is responsible for the leucocytosis, or increase of the white corpuscles, seen in inflammations and various infectious diseases.

4. Eosinophiles (Plates I., II., *f*). Cells resembling the polymorphonuclear variety in size and in the shape of the nucleus. The nucleus is usually stained less deeply than in the polymorphonuclear leucocytes. The cell is filled with coarse spherical granules, staining strongly with acid dyes, such as eosin or acid fuchsin. By Jenner's stain or with eosin they are a bright red, and with the "triacid" stain of Ehrlich they are of a purple or coppery-red color. They form from one-half to four per cent. of the total number of leucocytes.

5. Basophiles, or mast-cells (Plate I., *d*). Cells of about the same size as the eosinophiles and with a similar nucleus, staining feebly, as a rule. The cells are filled with intensely basophilic

granules, staining with methylene blue, thionine blue, dahlia, etc. By Jenner's stain they are usually purple, but do not stain by Ehrlich's triacid mixture, the basophile cells showing a pale, irregular nucleus and many white spots (the unstained granules). These are very scanty in normal blood, forming from one-fortieth to one-half per cent. of the total number of leucocytes.

In leucocythæmia the increase in the leucocytes is not shared equally by all these varieties, but in the lymphatic form the lymphocytes (large and small) form the bulk of the cells,—in the case from which Plate II., Fig. 2, was drawn the lymphocytes formed 99.2 per cent. of the total number of leucocytes,—whereas in the splenomyelogenic form these non-granular cells may be somewhat increased, as also the eosinophiles and the mast-cells, but in addition a new variety of cell is seen in the blood, known as the *myelocyte* (Plates I., II., *b*). This corpuscle is large, with a large pale nucleus, round, oval, or reniform, and frequently excentrically situated. The cell is filled with fine granules, neutrophilic or faintly oxyphilic in character, as in the polymorphonuclear leucocytes. Sometimes very small myelocytes, the "dwarf" myelocytes of Ehrlich, are seen, and also others with large spherical eosinophilic granules (eosinophilic myelocytes, Plate I., *e*). Myelocytes with basophilic granules have also been described, and one of these is seen in Plate I., *h*, stained by Jenner's stain. Sometimes cells intermediate between the polymorphonuclear leucocyte and the myelocyte are noticed (Plate I., *c*), in which the nucleus is rather more indented than is usual in myelocytes and not so contorted nor so deeply stained as in the polymorphonuclear leucocyte. Myelocytes may form as many as fifty per cent. of the total leucocytic count in a leucocythæmic blood.

In addition to these changes in the white blood-corpuscles in leucocythæmia, the red corpuscles may also be abnormal, the most common deviation from the normal condition (apart from deficiency of hæmoglobin) being the presence of nucleated red corpuscles,—generally normoblasts (Plates I., II., *i*), rarely megaloblasts. Another abnormality is degeneration of the protoplasm, as evidenced by unusual staining reactions in the red corpuscles affected. Instead of being pink or yellow in color, according to the stain used, the protoplasm is purplish red or brown, as shown in the plates. This is sometimes called "polychromatophilia." The nucleus in

PLATE I.

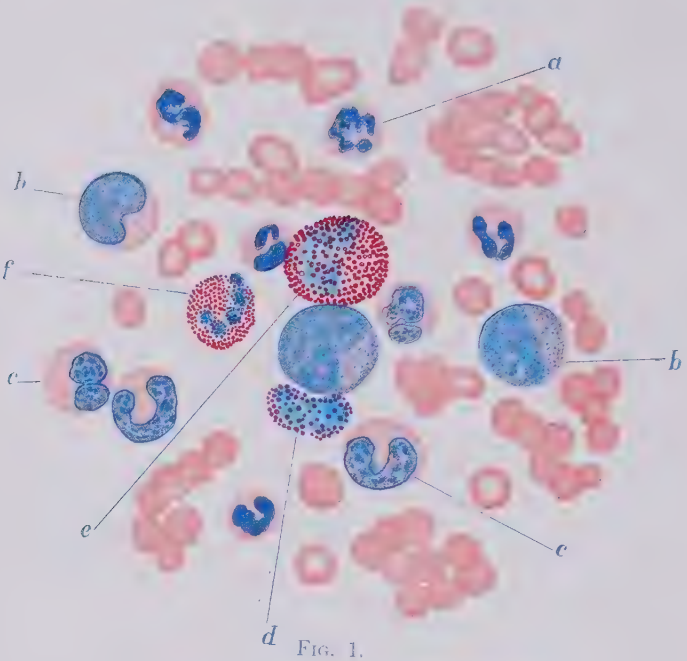


FIG. 1.

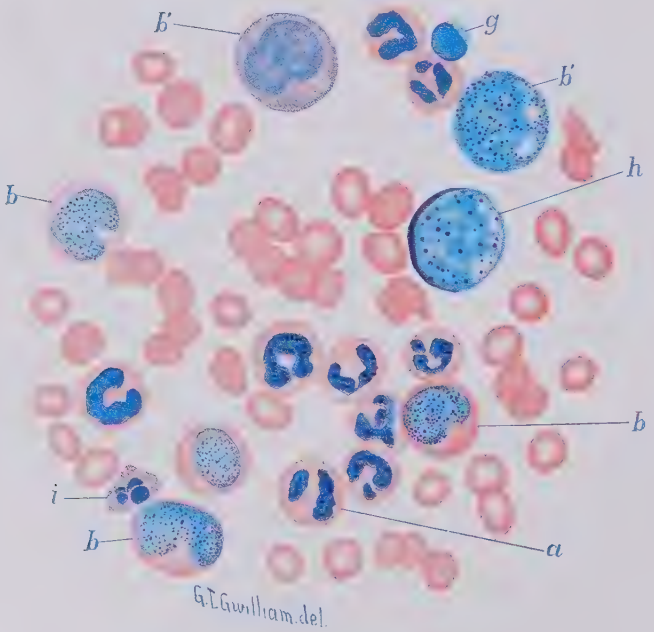


FIG. 2.

DESCRIPTION OF FIGURES.

PLATE I., FIG. 1.—Blood film of a patient suffering from splenomyeologenic leucocythæmia, stained by Jenner's stain. *a*, polymorphonuclear leucocyte; *b*, myelocyte with ordinary granulation; *c*, cells transitional between polymorphonuclear leucocytes and the myelocytes; *d*, basophilic leucocyte; *e*, myelocyte with eosinophilic granulation; *f*, ordinary eosinophilic leucocyte.

PLATE I., FIG. 2.—Blood film of same patient, stained as before. *b'*, myelocytes with granulation transitional between basophilic and ordinary neutrophilic; *g*, small lymphocyte or mononuclear leucocyte; *h*, myelocyte with basophile granules; *i*, nucleated red corpuscles, the nucleus being divided and the protoplasm stained purple (polychromatophilia). Other letters as in Fig. 1.

The figures were drawn from actual fields and magnified about one thousand diameters (obj. $\frac{1}{12}''$, oc. no. 4).

DESCRIPTION OF FIGURES.

PLATE II., FIG. 1.—Blood film of same patient as Plate I., stained by Ehrlich's triacid mixture. *a*, polymorphonuclear leucocyte; *b*, myelocyte with ordinary granules; *f*, eosinophilic leucocyte; *g*, small lymphocyte; *i*, nucleated red corpuscles (normoblasts), the protoplasm showing polychromatophilia; *k*, large lymphocyte or large mononuclear leucocyte.

PLATE II., FIG. 2.—Blood film of a child who died from acute lymphatic leucocythæmia, stained as Fig. 1. All the leucocytes seen in this field are lymphocytes (small (*g*), and large (*k*)), except for one polymorphonuclear leucocyte (*a*) in the upper part of the field. Of four thousand leucocytes counted, three thousand nine hundred and sixty-nine, or 99.2 per cent., were lymphocytes, only nineteen polymorphonuclear leucocytes, five eosinophiles, and seven myelocytes.

The figures were drawn from actual fields and magnified about one thousand diameters (obj. $\frac{1}{12}''$, oc. no. 4).

PLATE II.

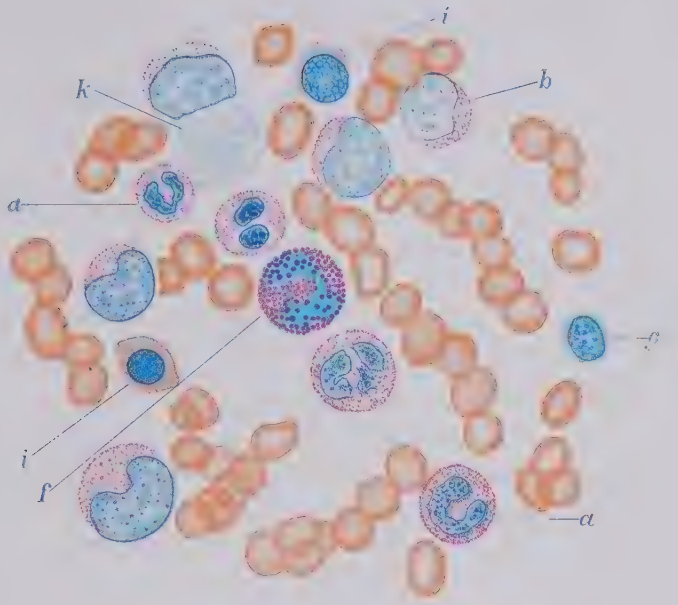


FIG. 1.

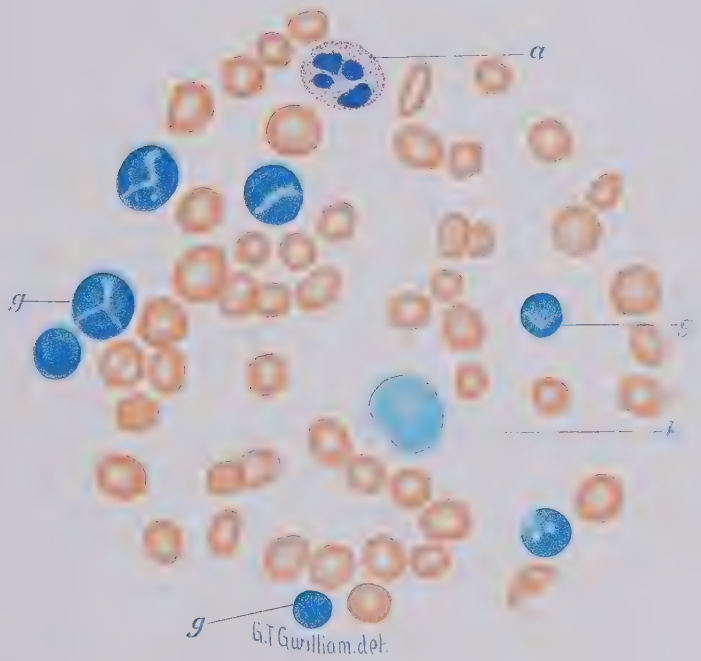


FIG. 2.

the erythroblasts is usually single and stains deeply; it may also be multiple (Plate I., Fig. 2), and in the "megaloblasts" often stains very feebly.

As a result of the examination of a large number of cases of leucocythæmia, Cabot gives the following summary of the blood condition.

A. *Splenomyelogenic.*

1. Red corpuscles diminished to three millions; many nucleated forms.

2. White corpuscles about four hundred and fifty thousand (on an average), of which—

3. Myelocytes form about thirty per cent.

4. Average ratio of white to red one to seven.

B. *Lymphatic.*

1. Red corpuscles diminished to three millions or less; nucleated forms rare.

2. White corpuscles one hundred thousand or fewer, of which—

3. Lymphocytes form over ninety per cent.

4. Myelocytes and eosinophiles very scanty.

5. Average ratio of white to red one to thirty.

C. *Mixed forms* occasionally occur, combining the characteristics of each of the above.

The best stains for examining and studying films of blood are (1) Ehrlich's triacid mixture,[1] used for from ten to fifteen minutes, after previously "fixing" the film by heating to 150° C. for from five to ten minutes or by immersing for thirty minutes or more in a mixture of equal parts of alcohol and ether; and (2) Jenner's stain, which is poured onto the film *unfixed* and washed off in distilled water after from three to five minutes. Jenner's stain is made by dissolving the methylene-blue salt of eosin in absolute methyl alcohol. In practice this stain is extremely simple; no fixation is required, the methyl alcohol fixing the film while the staining is in progress. Other stains used are eosin and methylene blue separately, eosin and hæmatoxylin, carbol thionine blue, etc.

It may appear to some of you that these details concerning the different leucocytes found in the blood are of academic rather than of clinical interest and importance, but that such is not the case

[1] This contains methyl green, orange green, and acid fuchsin.

one or two examples will demonstrate. In the first place, many pathological conditions cause a "leucocytosis," or increase in the number of leucocytes in the blood; a small unopened subcutaneous abscess, for example, may cause a very appreciable leucocytosis. Formerly it was taught that any ratio under one white to fifty red was to be regarded as indicating a "leucocytosis," whereas more than one white to fifty red indicated leucocythæmia. This position is untenable from both points of view, for in some cases of "leucocytosis" there may be more than one white to fifty red, and, secondly, in leucocythæmia the proportion of white to red may be less than one to fifty. The diagnosis can be made with certainty only by examining stained films. In "leucocytosis" the increase is due mainly to the polymorphonuclear leucocytes and, to a small extent, sometimes to the eosinophiles, whereas if the lymphocytes be much increased, and especially if myelocytes (finely granular and coarsely granular) be present, the case is one of leucocythæmia.

A second instance in which staining films of the blood and a differential count of the leucocytes are important is in cases of slight enlargement of the spleen, with some pyrexia and indefinite general symptoms, and in which the blood shows no appreciable increase in the number of the leucocytes. An examination of the stained film, however, shows the presence of myelocytes and an alteration in the relative proportion of the other leucocytes. One occasionally sees remissions of this type during the progress of an ordinary case of leucocythæmia, and if the patient first came under our observation during such a remission (when frequently the spleen is much reduced in size), and we omitted to examine a stained blood film, we should make an erroneous diagnosis and fail to detect a serious disease which might materially affect our prognosis.

Mr. S., aged twenty-three, went to Vancouver in 1899, considering himself to be then in good health. During April and May, which were wet and cold months, he felt very tired. In July he was attacked by "chills;" the rigors came on about midday, lasted an hour, then for an hour he felt hot in the head, with a final stage of perspiration. He was confined to bed for four days. Not regaining his strength, he returned to England in October, 1899. Late in August he had noticed that he was getting corpulent and thought he was getting fat. The abdominal enlargement, however, was found to be due to enlarged spleen when the patient was first

examined by Dr. Perram on December 13, 1899. Mr. S. first consulted Dr. Pybus on July 5, 1899, and again on July 11. He then complained of feeling ill, had a quick pulse and furred tongue, with a temperature of 104° F. Under antipyretics and quinine the temperature came down to 99.5° F., and on July 26 he was considered cured. During August and September he was subject to sensations of chilliness, but had no rise of temperature. When he left for home, on September 26, he seemed to Dr. Pybus to be suffering from some "pernicious toxæmia." Mr. S. told Dr. Pybus that while at Manitoba, about April 2, he stayed two days in a low, swampy place, and on July 20-27 he camped beside a small creek where there were swarms of mosquitoes. Dr. Pybus informs Dr. Thin that he saw three other cases of fever in a house two miles from that in which Mr. S. lived. These cases were similar in nature and were characterized by high temperature,—104° F. in one case and 106° F. in a second. The attacks in these cases lasted ten, sixteen, and twenty days respectively, and all made good recoveries. Dr. Pybus states that "these are the only cases of fever in the district during the ten years he has been in practice there, and were believed to be of a typhomalarial character. On March 28, 1900, Dr. Thin found Mr. S. very pale, anæmic, and with a "very large spleen, which descended to the groin on the left side and crossed nearly to the ilium on the right side." No malarial parasites or pigmented leucocytes were discovered on careful examination, but the white corpuscles were greatly increased in number.

On May 25, 1900, by the kindness of Dr. Thin, I saw Mr. S., and then made a complete blood examination. The spleen was as large as in March and was slightly tender about the notch. There was no enlargement of the liver or of any lymphatic glands. On September 8 I made another differential count of the leucocytes, and on November 16 I again saw Mr. S. and made a complete blood examination. The condition of the blood at the different examinations is shown in the subjoined table.

1900.	March 28	May 25.	Sept. 8.	Nov. 16.
Red corpuscles	. . .	3,250,000	. . .	2,966,700
White corpuscles	. . .	750,000	. . .	666,700
Ratio of white to red	. . .	1 : 4⅓	. . .	1 : 4½
Hæmoglobin	. . .	36–40%	. . .	40–42%
Color index	. . .	.6	. . .	.68
Nucleated red (per 1000 white counted)	13	51	10	20

The differential count is seen in percentages in the following table.

1900.	March 28	May 25.	Sept. 8.	Nov. 16.
Lymphocytes (small)	9 %	6 %	1.5 %	2.3 %
Lymphocytes (large)	10.6 %	4 %	2 %	.5 %
Polymorphonuclear leucocytes	41 %	47 %	38 %	38.4 %
Eosinophiles	10 %	8 %	8.6 %	10.8 %
Myelocytes	28 %	32.5 %	46.5 %	42 %
Basophile	1 %	2.5 %	.8 %	very few

Many transitional forms between large lymphocytes, polymorphonuclear leucocytes, and myelocytes were seen. The films were fixed by immersion in alcohol and ether, and stained by eosin and hæmatoxylin and by Ehrlich Biondi triple stain. In November Mr. S. was feeling so much better that he was desirous of taking up some occupation. The spleen was as large as ever, but causing no inconvenience beyond a feeling of weight when patient was tired. Treatment has benefited patient little as regards the blood condition or the spleen. He has at various times taken arsenic, iron, quinine, and preparations of hæmoglobin and bone marrow.

Remarks.—The interesting points in this case are (1) the antecedent history of malaria, almost certainly indicated by the "chills" described; (2) the great size of the spleen before attention was drawn to it and the slight inconvenience caused by it; (3) the great diminution in the percentage of lymphocytes (from twenty to three per cent.) and corresponding increase in the number of myelocytes (from twenty-eight to forty-two per cent.) between March and November, 1900; (4) in spite of the marked anæmia and the large number of white cells in the blood, the patient is apparently in good general health, as well as anxious and able to follow an occupation.

TO WHAT EXTENT CAN GENERAL PRACTITIONERS MAKE USE OF THE NEWER DIAGNOSTIC METHODS?[1]

BY ADDISON S. THAYER, M.D.,

Visiting Physician, Maine General Hospital; Professor of Diseases of Children, Bowdoin College; Portland, Maine.

A GENERAL practitioner of medicine, fifty-five years old, is directing the education of his son, aged twenty-five, in the hope that in this instance acquired traits will prove to have been transmitted, and that the usefulness of his son in the general practice of medicine will exceed the measure of his own attainment. It has been decided that the younger man, at the end of his apprenticeship, is to settle in a locality remote from his father's field; and this decision has contributed to self-reliance—on the part of the father. In the careful thought which he has given to things worth while for his son to know and to know how to do, he has incidentally put a value upon knowledge and skill as applied to his own personal needs. The son is not to become the father's partner; and so the father is debarred from the comfort of saying to himself, "I will postpone attention to blood-counts and cultures and laboratory tests until my boy is ready to share my work."

Although this son is already twenty-five years old, the elaborate education by college, medical school, hospitals, and perhaps travel, which his ambitious father has planned as affording the ideal equipment, is not yet complete. Facilities far better than the father enjoyed in his student-days are to widen the young man's knowledge of disease, are to train his organs of special sense to a prompt recognition of objective signs, and are to develop his judgment in weighing the value of symptoms, until he shall attain that power in rapid appreciation of salient points which passes for intuition and really becomes in a measure intuitive. This young man must know the secret exhilaration which a correct diagnosis can give.

An appraisal of diagnostic arts should fix a high rating upon

[1] Read at the annual meeting of the Maine Medical Association, June 5, 1902.

some of the methods of an earlier day. Amid the embarrassing riches of modern resource, our elder doctor feels an ever-growing satisfaction in letting his sensory nerve currents travel along certain well-worn paths of conduction. In skin disease and in disease of the nervous system, inspection is still the clearest guide; but present-day doctors should see more and see better than their predecessors, because they should know better for what to look.

Skill in palpation,—abdominal, thoracic, vaginal, and rectal,—an educated touch, which has served our doctor well for many years, he craves for his son in the highest attainable measure, even in these days of exploratory incision, when the knife is expected to reveal to sight what once could be determined, if at all, only by the sense of touch. The son's forefinger must have an added talent: it must sweep the juvenile pharynx and let no guilty adenoid escape.

The father has long cherished a secret pride in the promptness with which he has recognized percussion-notes betokening pleuritic effusion, an area of consolidated lung, or the enlargement of a solid organ. His son's ear must become equally alert. Concerning fancy grades of râles and shadings of percussion-notes, which cut so large a figure in text-books of an earlier date and so small a figure in his own actual experience, the father finds himself sometimes avowedly sceptical, and sometimes content with the dignity of reticence which so kindly serves the need of an uninstructed man.

Grateful as he is to the stethoscope, he acknowledges debt only in less degree to the various 'scopes which aid the sense of sight.

To the laryngoscope he credits an early hint of aortic aneurism which a motionless vocal cord has given. The laryngoscope now and then has revealed to him a new growth of the coarser kind. That boy of his, however, must be able to detect in the lining of the larynx the beginnings of a tuberculous inflammation. He must become deft in the practical art of posterior rhinoscopy.

How much reward may he or his son expect for time spent in using the ophthalmoscope? He knows that Sir William Gowers, although not an ophthalmologist, is expert as an ophthalmoscopist. But Sir William Gowers is also president of a short-hand association, is the inventor of a hæmoglobinometer, and, like Briareus and Hopkinson Smith, is not an ordinary man. The ordinary man occasionally by use of the ophthalmoscope can detect on the retina

an albuminuric spot which looks like the setting sun, and by somewhat more frequent use can be tolerably sure that a disk is choked if the choking is sufficiently violent. The doctor expects his son to approach nearer than this to the standard of Sir William Gowers.

Urethroscopes and cystoscopes have found no places yet in our doctor's kit of tools. Their revelations are known to him only by hearsay and by the demonstrations of courteous colleagues. Such acquaintance he regards as far too meagre for the needs of his boy, who must become fairly familiar with the uses of all such diagnostic aids, and adept in handling the more familiar specula, which open to view the ear, nose, pharynx, vagina, and rectum. Happily, this boy was born so late that he has escaped the once-prevalent epidemic use of the vaginal speculum for purposes supposedly therapeutic.

Nowadays the scope of all 'scopes is almost coincident with the limitations of the microscope,—known, in the slang of the laboratory, as "the scope." Modern bacteriology and hæmatology have their home in the laboratory, and the household god of the laboratory is the microscope.

Our doctor, in his thirty years of practice, not being one of those physicians who keep their microscopes under glass cases in their waiting-rooms to serve as impressive ornaments, has found his microscope, if not a friendly divinity, at least a useful friend. More than once his personal use of centrifuge and microscope has promptly cleared away his doubts. But the pressure of bedside and office work, of a kind which could not be deputized easily, have forced him to rely in the main upon eyes younger than his own for such interrogation by the microscope as his duties to patients increasingly demand.

His personal experience with laboratory methods, however, has brought him into sympathy with the customs of colleagues of his own age who never use their microscopes at all or who have no microscopes to use. He has found that manipulations which are easy for a laboratory specialist, by reason of constant practice and freedom from interruption, become a radically different undertaking for the general practitioner, whose occasional need may induce him to essay on his own account these same simple-appearing tests. To find in readiness the proper apparatus, reagents, and stains, to do the work, and then to do the clearing up, without hitch or interruption, is almost as much an instance of good luck as to

give the word to a field of horses, with no false starts or scoring for position.

So far as he is open to the fault of the Pharisee, his guilt consists in criticising his brethren not for sins of omission alone, but because they commit themselves sometimes to careless interpretation of the work of others. They order an examination of urine, and then pore over a document which states the varieties of casts and the fractions of a grain of sulphates, omitting to consider the more significant observation, which the analyst cannot make, as to the whole amount of urine excreted in a given time. In the case of a suspected throat, they sometimes put implicit reliance upon the report of the bacteriologist, to the neglect of more important clinical signs which they themselves should see. Our doctor has survived a period when other tests were over-valued. He has seen patients disregard the death-warrant which albuminuria or a cardiac murmur was declared to be, and persistently remain alive; and he hopes the day is at hand when no child will die for want of antitoxin because the report from a culture has not come in, and when no derision will be caused by a positive report of diphtheria concerning a person who does not know he is sick.

The zeal of his son in preparing microscopic slides of new growths and other tissues, like the craze of old-time doctors who collected botanical specimens, the father has found need to restrain and to direct, lest it become the passion of a curio-collector, rather than a step in the training of a therapeutist. His experience teaches that there are many tissues concerning which a general practitioner must have, in any case, the opinion of a well-trained pathologist, to whom the preparation of the specimen as a whole might well be left.

Short-cuts in technic, which have been evolved in the routine work of clinical laboratories, have already made available for the individual worker the use of many practical tests. Such are the rapid separation and speedy microscopic examination of urinary sediment and the staining and examination of sputum and of pus for tubercle bacilli. Short as the half hour may be which these procedures demand, it may happen to the man in general practice that this identical half hour is pre-empted beyond release.

And so the doctor's personal employment of laboratory tests has come to be limited, for the most part, to those which are simple

and speedy. Nevertheless, that son of his must become, on short notice, as ready with his knowledge and his tools to make a laboratory test as a surgeon is to make an amputation or a tracheotomy.

The new science of hæmatology, clinically considered, opens a puzzling problem in expediency. The doctor hopes that his son's activity in general practice will soon be such as to preclude him from making complete blood-examinations, as now conducted, except in cases where their value is obvious and where there is no one else to shoulder the task. Such an examination—including a study of the unstained blood, a red count, a white count, a differential count, and an estimation of hæmoglobin—is done rapidly if completed in two hours' time, while four hours would be a moderate allowance for such an examination in private practice.

He hopes, however, for time-saving devices which will make unabridged reports upon the blood available outside of hospitals, where now the work is done, for the most part, by internes and by senior students. Already in the estimation of hæmoglobin von Fleisch's instrument is being replaced by the simpler and equally accurate invention of Dare, and by the still simpler scala of Tallqvist, the use of which is the work of a moment. So, too, in the differential count, the sometimes vexatious fixing-process which Ehrlich's stain requires is yielding to the practical methods of Jenner and of Goldhorn.

In spite of pronouncements by well-known surgeons who disparage the value of blood examinations, the doctor knows that a leucocyte count may tip the balance between appendicitis and typhoid, and tip it in the way that is right. A few minutes spent in examining blood may positively identify a previously doubtful case of malaria. All this the doctor's boy must surely be able to do.

He must know the principles of practical bacteriology, how to make cultures and culture-media, and how to recognize the commoner pathogenic organisms. In determining the presence of the diphtheria bacillus, he must be so trained as to reduce to a minimum the mistakes of the laboratory. Whether or not, as a general practitioner, he is to be able to keep on hand fresh cultures of the typhoid bacillus for making Widal tests, he must know how to make such tests; and, to know how, he must have made them repeatedly himself.

For an ideal present-day equipment, he should know something

of the methods and effects of inoculating lower animals. He should have trained his eyes to the use of the fluoroscope, and his temper to the use of machines for producing Röntgen rays.

It is becoming plain that this ambitious parent has suffered a loss of perspective. In the rapid development of medicine during recent years, he has crowded into his boy's future programme one number after another until he expects a performance of which no finite being is capable; and he has developed in himself a species of mild megalomania. Although he claims to expect from the young man no more than a rudimentary grade of skill, this claim is palpable self-deception. He really expects the equal of his own keen stethoscopic ear to be straightway reproduced in his son, and, in addition, a fluoroscopic eye and various other twentieth-century accomplishments. He looks for a composite of the technical skill of the specialties of modern medicine to have the clearness of definition which is characteristic of each specialty.

He himself has never made investment in the paraphernalia required for radioscopy nor for practical bacteriology. For these aids in diagnosis he has relied upon experts. And here, if he did but know it, is the key to the puzzle. Believing as he does, with all his soul, that the function of the family doctor will never be lost, he should recognize also that this is distinctively the era of the expert; that the specialist should be a helper, not a rival; and that the helpfulness of specialists is notably promoted when family physicians know enough themselves to protect their patrons from fads and to tell them when and where to seek the benefit of special skill.

The general practitioner of the future should be more than a guide-post, more than a gatherer of urine, sputum, and tonsillar mucus. He should be, in the widest sense, a medical adviser.

MEANS OF TELLING WHETHER AN ATTACK OF SEROFIBRINOUS PLEURISY IS TUBERCULOUS.

CLINICAL LECTURE DELIVERED AT THE HÔTEL DIEU.

BY G. DIEULAFOY, M.D.,

Professor in the Paris Faculty of Medicine.

Gentlemen,—In the course of your medical career you will often be called to attend patients who while apparently in perfect health have been seized with acute serofibrinous pleurisy. The attack will present every appearance of an ordinary acute form of pleurisy, and its evolution will be that of a simple case. The conditions under which it occurred will seem to point to pleurisy *a frigore*. But you must question whether the disorder be as harmless as it seems. May not this apparently simple case be of a tubercular nature, making a great difference in prognosis?

The question, then, is how to solve this problem, frequently so difficult. Do we possess the means to enable us to diagnose exactly the different kinds of pleurisy? Is it in our power to assert whether an acute attack of pleurisy *a frigore* is tubercular or not?

At the time when I began my medical studies acute pleurisy was divided into primitive and secondary. The former was thought to result from cold, hence the term *a frigore*. It was also called idiopathic,—that is, not derived from any other morbid process. Furthermore, it was called acute pleurisy, which excluded the idea of any anterior disease in the patient. In short, the disease was simply called "pleurisy," without any qualifying term.

To this primitive pleurisy were opposed those forms which occur during the course of some other complaint, as pneumonia, rheumatism, Bright's disease, etc.; or those which have their origin in lesions of the neighboring organs of the thorax and abdomen. In this group of secondary forms of the disease tubercular pleurisy occupied first place; but the idea of a primitive pleuro-tuberculosis was not yet generally accepted, tubercular pleurisy being regarded as a complication of pulmonary tuberculosis.

Gradually, however, it was discovered that the forms of tuber-

cular pleurisy were very dissimilar. In the first list are now classed the cases that occur in persons suffering from pulmonary tuberculosis already in a more or less advanced stage. Occurring under these circumstances, that the pleurisy is tubercular is almost a foregone conclusion.

In a second list we will class those cases in which, while we are not sure regarding the tubercular origin, the presumption is warranted, even though the lungs be sound. Some of the patients have come of tubercular parents, for instance; others have had previously a tenacious attack of bronchitis, now well; or, perhaps, a single occurrence of hæmoptysis, or a fistula of the anus, successfully operated upon; or, possibly, a so-called attack of rheumatism which was really a tubercular attack of pseudo-rheumatism, the nature of which was not recognized. Others still bear distinct traces of suppurated glands of the neck in youth; or hip-disease that has left a slight defect in the walk; or adenoid hypertrophy with mitigated tuberculosis of the tonsils. In a word, the cases in this class have been preceded at a more or less remote period by tubercular localizations of which they are the outcome. Their evolution is not always that of acute pleurisy, and they occur after a preliminary state, of uncertain duration, characterized by languor and loss of flesh. Clinically, such cases are open to the suspicion of being tubercular.

The forms of tubercular pleurisy, however, of which I am now about to speak are quite different. Nothing either in the past or present points to tuberculosis. In perfect health and without appreciable cause, the patient is seized with pleurisy. Or perhaps it follows taking cold, as would tonsillitis or coryza. The beginning and course of the disorder, the evolution of the effusion, the result of thoracocentesis, when performed, and even the period of convalescence, are similar to what occurs in an acute inflammatory process.

This is the exact type of the so-called primitive pleurisy *a frigore,* about which you may be deceived, since several years later you may find the patient, who recovered entirely from pleurisy, suffering from pulmonary tuberculosis, the pleuritic attack having been the unrecognized initial manifestation of the tubercular infection. Another patient recovers entirely from pleurisy, only to succumb later to tubercular meningitis. Do not fancy that these are merely

coincidences or that the cases are rare. On the contrary, they are so frequent as to have awakened attention and to cast doubt upon the existence of simple pleurisy *a frigore.*

In 1884 Landouzy took the initiative and boldly declared that all cases of pleurisy the origin of which could not be discovered were tubercular, no matter how robust might be the patient. Other physicians sustained this view, and opinion set so strongly in that direction that pleurisy *a frigore* lost much of its prestige.

Still, Landouzy's declaration awakened much protest. It was regarded as exaggerated, and opposing facts were produced in evidence. There were published many cases of patients who had completely recovered from acute pleurisy *a frigore* which was manifestly not tubercular, since the disorder had not been followed, either soon or later, by the tubercular manifestations. The medical world was divided into two factions equally convinced of the truth of their views as to the existence of a true pleurisy *a frigore.*

It remains for us to-day to decide for ourselves what is the truth in this matter, as it is of great importance to be able to class the two types correctly.

Let us first see what information can be derived from the pathological anatomy of the case. It is by no means rare for sudden death to occur in a case of pleurisy with abundant effusion. In my own limited researches, I have found about forty cases of this kind, and you can readily infer that they are relatively frequent. In some of the cases cited, where the patients had been considered true types of pleurisy *a frigore,* post-mortem examination revealed tubercular lesions of one or both lungs, foci which had been absolutely latent during life. In other cases the evidence of tuberculosis was found in the pleural membrane and had also remained unsuspected.

In yet other cases patients, apparently completely recovering their health after an acute attack of pleurisy, are a few weeks later suddenly carried off by acute miliary tuberculosis or meningitis. In 1884 a vigorous man of twenty-eight, a mechanic, came into my wards for acute pleurisy *a frigore* that began three weeks previous. There was no cause to suspect tuberculosis. I found a large effusion of about two litres on the left side. Of this I drew off one-half at once and the rest after a few days. In due time the patient apparently recovered perfect health. Six months later,

however, he returned to the ward and died in a few days. *Post-mortem* revealed the pleurisy healed, leaving only a few adhesions; but there were tubercular meningitis and granulations in the lung as well.

In his anatomical and experimental researches, the conclusions reached by Péron are as follows: "So-called acute pleurisy is tubercular in an immense majority of cases, the tubercular infection of the pleural membranes being often but moderate."

Having noted the conclusion to which the *anatomical* data point, what information do we obtain from the *clinical* side of the question?

In many cases clinical teaching confirms the showing of pathological anatomy by demonstrating that patients are in reality already touched with tuberculosis previous to an acute pleurisy, since, although they completely recover from the latter, later the tubercular infection invades some portion of the body. It is therefore plain that with these persons the pleurisy was merely a more or less early localization of the bacilli.

But clinical experience also teaches that other patients in whom acute pleurisy with abundant effusion has healed without leaving any tubercular taint, have never shown at any later period any manifestation of tuberculosis. Nor was it manifested in their children.

In regard to this matter several statistics have been published. That by Fiedler, in Germany, comprises ninety-two cases of serofibrinous pleurisy in which thoracocentesis was performed. Of this number seventeen died of consumption in the hospital, eight met the same fate after leaving the institution, and sixty-six left the hospital tubercular or suspected of tuberculosis, while twenty-one remained well after at least a year.

Other statistics published by Barrs, Bowditch, Mayr, and Ricochon show results that do not all agree. Nevertheless, the tubercular element largely predominates.

A few years ago, at my request, a number of my colleagues at the faculty gave me the result of their experience. M. Vergely sent me four histories concerning cases of pleurisy where he had aspirated from fifteen to twenty-two years before, which had remained perfectly healthy, both patients and offspring. M. Lépine cited a half-dozen cases treated many years before which had never

been followed by tubercular manifestations. M. Dreyfus-Brisac had seen a number of patients remain in perfect health after recovering from acute pleurisy, and M. Coriveaud has published an interesting paper concerning the very favorable nature of many cases which he treated during a period of twenty-five years.

During the last thirty years I have performed thoracocentesis in a great many cases of pleurisy. I have lost sight of nearly all my hospital patients, but have followed closely a number of private cases, of which eleven of acute pleurisy requiring aspiration fully recovered without any later manifestation of tuberculosis.

M. Lamarre furnished the following interesting facts. In former times the supply of hay for the cavalry regiment at St. Germain was nearly a mile from the barracks. At about the half-way point was a three-foot wall around the old castle. Here the soldiers rested with their burden of one hundred and ten pounds of straw and hay. They were usually perspiring freely when they reached the wall, and leaned against it so that their load would lie on top of it. M. Lamarre, then assistant physician to the hospital, noticed that an epidemic of acute pleurisy *a frigore* attacked the regiment, while civilians went free. All these attacks were on the right side, and the patients recovered perfectly, with or without aspiration. (I should have stated that the wall had been but recently cut down to its convenient height.)

The cases were unquestionably pleurisy *a frigore,* says M. Lamarre. The corner of the wall where the perspiring soldiers rested was sheltered against every wind except an icy one from the northeast, which blew directly upon the right side of the men. So unmistakable was the evidence of the action of cold in these cases that the men were forbidden to rest at the wall, and the epidemic ceased.

But in this barrack, the regiments changing every six months, each new one fell a victim to the epidemic of pleurisy, until several had gone through the experience. In each case, forbidding the resting corrected the evil. In all there were about fifty cases arising from this cause, none, however, proving fatal.

In addition to these rather special cases cited, M. Lamarre has seen during the last thirty years about twenty cases in his private practice where recovery was not followed by ulterior tuberculosis.

Clinical facts, then, would seem to prove that recovery from acute pleurisy without ulterior tuberculosis is not so very unusual, these cases furnishing an argument in favor of the production of that disorder by cold. It is contended also that as pleurisy *a frigore* manifestly exists in the lower animals, why not, therefore, in man? M. Trasbot has demonstrated that pleurisy *a frigore* is common in horses, dogs, and sheep, where it may have no relation with tuberculosis. In support of this view he cites a case of a regiment of cuirassiers, hastily formed during the war of 1870, where the horses were freshly bought and had their hair clipped. These animals being kept out of doors, and frequently without any covering, in a few weeks' time there had developed among them thirty cases of pleurisy, almost all proving fatal. The result was that no more sheared horses were purchased.

Another veterinary surgeon saw one hundred cases of pleurisy, sixty proving fatal, in a flock of four hundred sheep that had been sheared during a very cold February. These animals were not tubercular, the liquid aspirated and injected into guinea-pigs not producing tuberculosis.

Another observer saw several very vigorous dogs taken with pleurisy after having followed a stag in a pond for nearly an hour in midwinter. None of these animals were tubercular.

Says M. Trasbot upon this subject, "The three domestic animals—horse, dog, and sheep—in which pleurisy is most often found are just those least disposed to tuberculosis. Thus the facts acquired through clinical observation among several animal species are in direct opposition to the idea that pleurisy among those species is a form of tuberculosis. An additional fact, not only deduced from clinical teaching but absolutely proved by experiment, is that tuberculosis has never been contracted by guinea-pigs or rabbits through injections of the serofibrinous pleuritic effusion of the horse or dog."

It is therefore certain that non-tubercular pleurisy *a frigore* is common among animals; but as we are only concerned with human pathology, we must decide whether with man such a thing really exists, and, if so, how it may be differentiated from the tubercular form.

The information must be sought in the modern laboratory.

The injection of pleuritic liquid into the peritoneum of guinea-

pigs has furnished valuable data. MM. Chaffard and Gombault obtained in this way positive results in half of their cases; in twenty cases, inoculations made by M. Netter produced eight positive results of tuberculosis. All going to prove that, in a number of cases the disorder was really tubercular though seemingly but primitive *a frigore*. Later, the same author published a series of fourteen cases, of which five appeared to be primitive. Two of the latter transmitted tuberculosis to the guinea-pig. When the peritoneum of the guinea-pig is inoculated with twenty cubic centimetres of the pleural liquid, instead of a small dose, the chances of success increase. Even then the experiment often gives no result.

In other words, the value of inoculation is absolute when the result is positive, but a negative result does not prove that the attack is not tubercular. Every observer has found that the injection of pleural liquid from a case that is manifestly tubercular may be followed by a negative result. The reason is obvious. The effusion being only slightly infected, the bacilli are thinly scattered through the pleural liquid, and but few of them enter the animal's peritoneum, where they are rapidly destroyed.

Thus, while inoculation is surely an aid in investigation, it is not infallible.

Injections of tuberculin supply information, the value of which cannot be disputed. According to reports published by the German clinics, patients seized with pleurisy of an apparently simple form, but which is in reality tubercular, are almost as sensitive to injections of tuberculin as are those who are manifestly tubercular. But utmost caution must be exercised in making these injections. The patient's temperature must be normal. If the rectal temperature, taken four times in twenty-four hours, shows absolute apyrexia, then, and then only, should the tuberculin test be attempted.

The amount of tuberculin to be injected varies from a tenth to a quarter of a milligramme, diluted in sterilized water. If the patient be tubercular, the reaction and rise of temperature begin in twelve to twenty-four hours after the injection. It may happen, however, that the dose injected is too small to give rise to the expected reaction with a given patient, or that a slight reaction may occur where there are no tubercles. A few years ago I made some injections of very small doses of tuberculin, with results so unsatis-

factory that I abandoned the method. In veterinary practice, however, the results are excellent.

It is only recently that the use of the culture method for the detection of the tubercle bacillus in effusions has been successfully tried. For positive results it is necessary to have a culture medium particularly suited to Koch's bacillus, such as a mixture of blood, gelose, and glycerin. Rabbit blood, extracted aseptically, mixed with gelose, gives a culture medium on which will freely develop microbes that do not grow on ordinary media. And if glycerin is first added to the agar-agar, there is obtained this preparation of gelose, blood, and glycerin, on the surface of which it is only necessary to deposit the suspected liquid, whether it be spinal or pleural liquid or pus.

Instead of ordinary culture tubes, small Erlenmeyer receptacles may be used. In the bottom a layer of the medium is allowed to settle. In this way a much larger quantity of the liquid under examination may be spread over the surface. The receptacles are then carefully covered and placed in an oven at 37° C. In three or four weeks it will be found that several colonies are developing, and that they increase in number. In the beginning they are smaller than a pin's head, but they soon grow larger, extending by means of chocolate-colored, spherical granulations. They are less difficult to dissociate than are tubercular colonies that have developed on other media. Under the microscope, the preparations show bacilli, single or in groups, arranged in the manner habitual to them. The number of colonies detected by the eye depends upon the amount of effusion inoculated, and especially on the number of bacilli contained in the effusion.

This method of cultivating the bacillus as a diagnosis-test of pleural effusion is valuable, since to detect in this way the presence of the bacillus and to see it with one's own eyes would be conclusive proof were it more reliable. The reason that this test has not proved more successful among our own patients is probably that the liquid of many cases of tubercular pleurisy is only slightly virulent, much less so than that of the tubercular effusions—of the meninges, for instance. In our experiments but one tube was used; but the chances of success would be undoubtedly multiplied were a number inoculated with specimens of the pleural liquid taken from different points of the effusion.

We now come to the serodiagnosis and its connection with tuberculosis. Broadly, any serodiagnosis presupposes two factors, no matter what may be the disease. On the one hand must be a homogeneous culture in a liquid medium, with the microbes living apart from each other; and on the other hand must be some of the patient's serum, the mixture of which with the culture produces agglutination of the microbes.

In 1898, M. Arloing determined to find for tuberculosis a means of serodiagnosis comparable to that found by M. Widal for typhoid fever. He solved the problem by obtaining homogeneous cultures of Koch's bacillus in a liquid medium.

To insure the greatest chance of success your culture of tubercle bacilli should be twelve days old. A little of the culture should be placed in a tube of small size and caliber. With this culture should be mixed some blood serum taken from the finger by means of a lancet, using one part of serum to either five, ten, or twenty parts of culture. The tube should then be shaken, in order that the mixing may be thorough. If the result be positive, agglutination takes place in from one to five hours. The upper portion of the liquid becomes clear, while at the bottom of the tube will be an accumulation of flakes which render the liquid opaque. Microscopical examination shows the bacilli united in groups.

Many cases of pulmonary tuberculosis begin in an almost latent form, and cannot be detected at once by the usual means employed; the diagnosis of tubercular peritonitis with ascites is often uncertain; and tuberculosis of the bones and joints is often specially difficult to detect in that it may assume the form of acute arthritis, dry or chronic arthritis, or even acute articular rheumatism. Under such circumstances, the serum test, by defining the tubercular nature of the lesions, has shown the error of first diagnosis.

Other writers have drawn conclusions that differ somewhat from the foregoing, but it is absolutely necessary to success that the cultures should be of the proper age; furthermore, it must be remembered that in tuberculosis the agglutination scale is not so extensive as in typhoid fever.

In experimenting with serodiagnosis of tuberculosis as applied to pleurisy, M. Courmont says that a positive sero-reaction may be obtained by mixing with the culture of Koch's bacilli either the patient's blood serum or the pleural liquid in the proportions

already given. The agglutinating power of the blood is not always the same as that of the serum. It may be greater or less, and may exist although the pleural liquid fails, and *vice versa*. Positive sero-reaction is more unusual when the pleurisy is associated with serious lesions; on the other hand, it is more common in mild, incurable cases of tubercular pleurisy. Pleural reaction may be absent in the earlier stages of pleurisy, and only become positive after a certain length of time, usually towards recovery. With the pleural liquid M. Courmont has obtained positive results in twenty-three cases out of thirty-one; with blood serum, in eighteen out of twenty-two.

This demonstrates that the serodiagnosis applied to pleurisy *a frigore* suspected of being tubercular is an excellent test.

It was thought that Ehrlich's diazo-reaction might give useful information relative to the diagnosis of tubercular pleurisy. Though positive results have been obtained in tubercular cases, the method is too uncertain to be relied upon to solve the problem on which we are at work.

But a new means has recently been discovered, cytodiagnosis, which is based upon the quality of the cellular elements found in pleuritic effusions and in the effusions of serous membranes in general.

When the system is attacked by pathogenic agents of any sort, cellular elements reply by a reaction denoting resistance and defence; not always the same cells, however, as there is, according to the case, a species of cellular selection. Thus, the Metchnikoff's microphagic elements consume such microbes as the streptococcus or the pneumococcus. The large, mononuclear or macrophagic elements are more powerful and can absorb, and sometimes destroy, Koch's bacillus. They readily digest blood-globules and polynuclear cells. It was therefore natural to infer that the nature of the pathogenic element which had given rise to an illness might be determined by the variety of cells found in the pleural liquid. To this idea was due the discovery of the cytodiagnosis.

In 1900 MM. Widal and Ravaut published the first description of this method. To discover the cellular elements in the effusion of a case of pleurisy, draw off, with all aseptic precautions and with a sterilized syringe, a few grammes of the liquid; drop these into a test-tube and centrifugate. A deposit will then be found at the

bottom of the tube. Decant so as to leave only a little liquid in the tube, which will form a cloudy emulsion with the deposit at the bottom. By means of a platinum wire put a drop of this emulsion on a slide and color with thionine, eosin, hæmatein, or Ehrlich's triacid.

Under the microscope these preparations will present a variety of forms of cellular elements,—blood-globules, polynuclear leucocytes, large mononuclear cells, lymphocytes, and endothelial cells, either separated or united in flakes. But all these various elements are not present in every case of pleurisy. A given case will not contain at one time nor in any amount polynuclear cells, lymphocytes, and endothelial cells.

Setting aside the red globules which are found in greater or less amounts in nearly all pleural effusions, a given sample will contain almost nothing but lymphocytes, the polynuclear and the endothelial cells being absent or in very small numbers. Another sample will contain almost nothing but polynuclear elements, the lymphocytes and endothelial cells being wanting, or practically so. And finally, yet another sample will contain nothing but endothelial cells, separated or united, the lymphocytes and polynuclear elements being practically absent.

This preponderance (sometimes almost exclusive) of a certain cellular element in a pleuritic effusion constitutes what is called the cellular formula of that effusion and gives the cytodiagnosis. Thus, there are three kinds of pleurisy.

A first variety concerns the pleuritic effusion of heart disease, Bright's disease, and cancer, as well as of effusion due to neighboring irritation or compression. In such cases, infectious agents being absent, there is no cellular struggle; nothing but the partly mechanical acts of transudation and desquamation, a sort of congestive œdema, the liquid taking with it the endothelial cells of the serous membrane as it passes through into the pleural cavity. In such cases, the liquid generally contains neither lymphocytes nor polynuclear elements, at least in the early stages. Examination shows almost nothing but large endothelial cells coming from the desquamation of the serous membrane.

On a preparation of this sort the number of the endothelial cells will be found very large as compared with the red globules and leucocytes; they are isolated or in groups, sometimes in large patches

or flakes, with polycyclic edges. After coloration their nuclei are seen to be much darker than their protoplasm; the contour of the cells is nearly circular.

These endothelial flakes are characteristic of mechanical pleurisy. MM. Widal and Ravaut found them in twelve cases of this sort, and the post-mortem of three of the patients, as well as the negative results of intraperitoneal inoculations with guinea-pigs, made with the liquid taken from seven other cases, shows that in such circumstances tuberculosis is not present.

These facts may be of great value in clinical medicine. For instance, any pleuritic patient who has had hæmoptysis is naturally open to the suspicion of being tubercular. But hæmoptysis is not rare in heart disease, and such a patient can also have pleural effusion. The cytodiagnosis will reveal at once that the pleural liquid with its endothelial flakes is of mechanical effusion and not of tubercular pleurisy, a matter of moment to the patient.

Recently, we discussed two cases of hæmoptysis in Bright's disease, a complaint which predisposes to hemorrhage of the bronchial tubes and nose; and you then learned that this hæmoptysis may appear before the nephritis has manifested itself by any serious symptom. If with such a patient there occurs a pleural effusion, knowing the previous hæmoptysis, you may at once think of tubercular pleurisy. Whereas, if upon examination the liquid reveal the endothelial flakes, it will be proved that the effusion is mechanical and not one of tubercular pleurisy.

While it is true that in the first stages of the disease lymphocytes do not appear in these mechanical effusions with endothelial flakes, later on they may become numerous. Nevertheless, the presence of the flakes will settle the diagnosis.

In the second variety of pleurisy the cellular formula is quite different. Here we have acute, infectious pleurisy where the pathogenic agents, streptococcus, pneumococcus, typhoid bacillus, etc., after having given rise to cellular reactions of resistance and defence, may themselves be no longer present in the liquid. But the evidences of the struggle, phagocytes or polynuclear leucocytes, may be there in great numbers. There will also be found large, mononuclear cells, which are at times large leucocytes. At others, they originate in the serous membrane, as is sometimes demonstrated by the presence of isolated, at times normal, endothelial cells.

In short, the polynuclear and the mononuclear elements characterize acute infectious pleurisy; the lymphocytes, when they are present, are quite a secondary matter, and only exceptionally will be found any small endothelial flakes with two or three nuclei.

In regard to this variety of pleurisy MM. Widal and Ravaut present the following: "Three times out of seven, in typhoid pleurisy, the relative abundance of the large polynuclear leucocytes characterized the formula of the effusion. In a case of streptococcic serofibrinous pleurisy there were only polynuclear elements with deformed nuclei. The liquid of pneumococcic, serofibrinous pleurisy is characterized by the presence of red globules and of some lymphocytes. But especially is there an abundance of polynuclear elements and a considerable number of large mononuclear cells, of which some have absorbed the polynuclear cells into their protoplasm."

These large elements are at times big, mononuclear leucocytes; at others, endothelial cells, originating in the serous membrane. It is only exceptionally that there are any endothelial flakes with two or three elements.

We now come to the most important of all, the third variety of pleurisy, which corresponds most nearly to acute pleurisy *a frigore*. Under the microscope preparations from the liquid of this variety show that its cellular formula is characterized by the almost exclusive presence of lymphocytes, very close together and mixed with a relatively large number of red globules. Sometimes a mononuclear cell will be found here and there. The polynuclear elements, if present, are very few, and are probably due to secondary infection. Endothelial flakes must be extremely rare, as in the seventeen cases examined by MM. Widal and Ravaut they were entirely absent.

This cellular formula, pleural lymphocytosis with red globules, declares at once that a case of pleurisy is tubercular; and the histological examination of the pleural membrane, as well as inoculating the peritoneum of guinea-pigs with the effusion of such a case, confirms the diagnosis of tuberculosis.

Of all the tests we have discussed, cytodiagnosis is much to be preferred. It is easy, rapid, and perfectly harmless. Its absolute accuracy only future experience will demonstrate. Cytoscopic examination is fully as necessary as is bacteriological analysis of the

sputum; and in the future any case history of pleurisy in which the cytodiagnosis does not appear will have to be considered unsatisfactory.

An attack of pleurisy which is regarded as tubercular because it appears in the course of manifest pulmonary tuberculosis may not be tubercular at all; while another attack, considered as due to influenza because it appears during the course of that disorder or its convalescence, may be a tubercular manifestation developed by the influenza infection. This has occurred in many cases, and certain attacks of serofibrinous pleurisy of traumatic origin, which might be ascribed to the trauma alone, are, in reality, tubercular, the trauma having only favored the appearance of the latent bacillary process.

We have so far considered only acute tubercular pleurisy, but the cellular formula appears to be applicable as well to cases several months old. We have recently had in the wards a man who had already been treated for this disorder for some length of time. Aspiration revealed a lemon-colored effusion; the cytodiagnosis gave the formula of tuberculosis; and lymphocytes, red globules, and the serodiagnosis, all pointed to the same conclusion.

But the formula may be quite different in chronic tubercular pleurisy, and particularly in tubercular hydropneumothorax, or when pleurisy arises from contact with advanced tubercular lesions of the lung. In such cases it is not the lymphocytosis that predominates. There will be found old, deformed polynuclear cells, with their nuclei much divided, as well as elements with vesicular protoplasm. Sometimes, even, there will be amorphous conglomerations that seem to have originated in the endothelium.

In this class of cases the diagnosis must be made with your clinical sense. If there still be doubt, recourse must be had to cultures, inoculations, and the serodiagnosis. But it must be remembered that where the lesions are chronic, the serodiagnosis and inoculations give less satisfactory results than in cases of those of more recent origin.

It is demonstrated, then, that with the means in our possession at the present day, we are in a position to form a fairly accurate opinion of the true nature of the disorder formerly known as pleurisy *a frigore*.

We note, in the first place, that the majority of such cases are

really tubercular. The cytodiagnosis, with its pleural lymphocytosis, reveals this fact.

Next, we consider that there are agents capable of giving rise to acute attacks of pleurisy which are in no way connected with tuberculosis. One person is attacked with serofibrinous pleurisy just as he would be attacked by pneumonia, the pneumococcus being the cause; a second has pleurisy with effusion, due this time to the streptococcus; a third has acute pleurisy with effusion, and fetid or putrid pleural infection is found.

These forms of acute pleurisy, with still others, have nothing to do with tuberculosis. Bacteriology classifies them according to their pathogenic agent, and the cytodiagnosis groups them under one cellular formula,—cases of pleurisy with polynuclear or mononuclear cells.

The pathogenesis of pleurisy, then, formerly so obscure, is now well understood; and the pathogenic diagnosis of its different forms has been so clearly elucidated that the question arises whether there shall be left in human pathology any phase at all for this essential pleurisy *a frigore,* formerly occupying such a prominent position.

Another side of the subject now comes under consideration. Having admitted that an attack of acute pleurisy *a frigore* that cannot be ascribed to any other cause is tubercular, how can we explain the fact that it often recovers without leaving any trace whatever?

To this it is replied that the fact that these cases recover does not prove that they are not tubercular. Tubercular pleurisy is sometimes a local and primitive form of tuberculosis of the pleura, the pleura alone being affected, the rest of the system remaining healthy. And since these serous membranes are well armed for defence, it is not surprising that the attack recovers without further spread of the infection.

Besides, curability is not peculiar to pleural tuberculosis. Tuberculosis of the articular synovial membranes, from simple arthritis up to tubercular pseudorheumatism, frequently recovers, and tuberculosis of the pericardium does the same. Cases of recovery from peritoneal tuberculosis, particularly with ascites, are innumerable. The attacks of ascites that were formerly termed essential, or *a frigore* are undoubtedly mild forms of tubercular peritonitis

that are easily cured. Some recover under medical treatment alone; others after simple puncture; and others, again, after laparotomy. This curability is also found in simultaneous tuberculosis of the pleura and peritoneum.

Tubercular pleurisy may, then, not only be regarded as curable, but at times perfectly harmless. M. Péron, who has studied this question from an anatomical and experimental stand-point, has drawn the same conclusions. "In cases of tubercular, serofibrinous pleurisy," says this writer, "manifesting the clinical appearance of acute tuberculosis, the bacillary infection is reduced to a minimum, whereas the total organic reaction is considerable."

Since the date, now distant, when I invented the method of aspirating for pleurisy, the operation has been performed many times in my wards, and for many years my internes published these cases in full detail. These documents prove that tubercular, pleural effusions, whether primitive or secondary, can be cured by aspiration. Further, I declare that when aspiration is properly performed recovery is almost the rule, particularly in cases where the tubercular pleurisy is apparently primitive.

In speaking of recovery, however, we desire to go further than the attack of the moment, and learn the ultimate result to the lungs. This is the whole question, and the reply is difficult. We have learned that complete recovery belongs particularly to primitive pleuro-tuberculosis, where the localization is purely pleural and the lung is sound. But how can we affirm that the lung is sound in a person who has an attack of pleural tuberculosis apparently primitive? We know that acute pleurisy is often accompanied by pulmonary congestion; and what is the nature of this congestion? A small focus of tuberculosis may exist in the lung and not reveal itself by the least symptom; yet this unsuspected focus may at a certain moment be the origin of a tubercular manifestation. Thus, an acute pleurisy soon carries off its victim that may have every appearance of a primitive disease, whereas post-mortem examination will disclose a small tubercular focus.

This also holds good for tubercular pleurisy. A person in good health is suddenly attacked with acute pleurisy; the cytodiagnosis, with its pleural lymphocytosis, proves the trouble to be tubercular; all the signs point to primitive and local pleuro-tuberculosis; and most minute examination shows no lesion of the lungs. All this,

and yet this pleuro-tuberculosis, apparently primitive, may probably be due to a small pulmonary focus that has contaminated the pleural membrane, having passed through an absolutely silent evolution. This all proves that really primitive pleuro-tuberculosis (the form that is associated with no pre-existing lesion of the lungs), and pleuro-tuberculosis due to contamination by a latent pulmonary focus, may both have every appearance of acute pleurisy *a frigore*. Clinically they may show no difference whatever, and the cytodiagnosis unites them in the same cellular formula,—*i.e.*, pleurisy with lymphocytes and red globules.

And yet, as regards prognosis, these two varieties of tubercular pleurisy are not to be compared, one being less serious than the other. With primitive tubercular pleurisy, the pleural lesion may recover without extending farther; but if the lung be already diseased, however slightly, the outlook for the future is less favorable, as one must treat a double tubercular lesion,—that of the pleural membrane and that of the lung. It will thus be seen that the ultimate prognosis of tubercular pleurisy is rather uncertain. And even with acute pleuro-tuberculosis, too much hope must not be built upon its relative mildness.

Acute pleuro-tuberculosis is usually accompanied by a large effusion. This effusion may possibly be a means of defence. It may be that the lung, forced into smaller dimensions by the volume of the effusion, is less likely to be invaded by the pleural tuberculosis. I am not prepared, however, to assert this as a fact. Should the hypothesis be true, it might be advantageous to delay aspiration a little. But, in opposition, we know how serious it is to permit too large an amount of liquid to accumulate in the pleural cavity, sudden death not infrequently resulting from such a course.

Earlier you were shown how frequent is this terrible accident. There was no difficulty in citing at least forty published cases. Naturally, many have not found their way into print. A complete list would probably show the number far greater than we had thought.

In looking over these case histories they seem almost identical. It is seen that the patient who died suddenly had an effusion that was causing a dyspnœa so insignificant that in many cases aspiration was not under consideration, or was imprudently deferred till the following day.

It must always be borne in mind that in pleuritic effusion the symptom dyspnœa is one that is very uncertain and that may deceive you altogether in deciding whether aspiration is urgent or not. The only sign for guidance in this matter is the volume of the effusion in the pleural cavity. Except in an extraordinary case, a patient does not die suddenly with less than fifteen hundred grammes in the pleural cavity, but if there be more, death may occur at any moment. It is necessary, then, to be able to make a fairly approximate estimate of the volume of pleural effusion. This is not difficult, thanks to the information to be derived from the percussion, the auscultation, and the displacement of the organs (notably that of the heart in pleurisy of the left side). Radiographic examination is far from giving us such exact indications.

Having determined its necessity, perform aspiration at once, being careful not to draw more than a litre at one time, which will prevent the liability of accident. You may find that in acute tubercular pleurisy the effusion recurs rapidly and abundantly after having been all drawn off by aspiration. It has been my experience that this rapid and persistent recurrence is much less common in cases of acute infectious pleurisy that are not tubercular. On several occasions in patients with acute tubercular pleurisy two or three litres of serofibrinous liquid, drawn off at two or three different times, seemed to exhaust the effusion. Its physical signs were replaced by rubbing sounds, and the patient was regarded as cured. But without fever, dyspnœa, or pain the pleural effusion gradually and insidiously recurred to such a degree as to attain in a few days one and one-half to two litres.

The consequence of such an occurrence may be readily seen. Having aspirated at the proper moment, you regard your patient as protected from danger; and his general condition seems so good that it is deemed unnecessary to see him every day, and you auscultate him less frequently. But suddenly the patient dies, and the post-mortem reveals the cause in a big effusion that has been gradually reforming.

Particular care, therefore, must be exercised in cases of acute tubercular pleurisy. It is treacherous and insidious, and the irregularity of its evolution is not the least interesting side of its history. Acquire the habit of estimating the volume of an effusion, and lose no time in aspirating as soon as it is deemed necessary.

Even after the acute stage seems over, the patient should be carefully watched and examined every day; for it must be borne in mind that the liquid that has been removed may reform rapidly, and sudden death ensue if you are not watchful.

The advice just given concerns the active phase of acute tubercular pleurisy, but the treatment naturally does not end here. The patient is convalescent, but beneath the appearance of recovery lies the tubercular lesion of the pleura, and what there is in store for him is a question. Will the pleurisy recover without leaving any trace, or is it merely the first stage of a tubercular infection which, sooner or later, will attack the lung or some other organ? When the pleura was attacked, was the lung healthy or did it contain an unsuspected tubercular focus? These points are by no means clear, but one thing is certain, and that is that the patient is tubercular and should be placed in the most advantageous hygienic and therapeutic conditions.

To accomplish this a convalescent from acute tubercular pleurisy should be made to understand that extreme care upon his own part is absolutely necessary, even if he considers himself absolutely cured of the active phase of his attack. The spread of the tuberculosis to other organs is always a possibility. Hygienic means are the ones that will insure the best results, and all causes that tend to the overwork or the running down of the system should be avoided. Food should be substantial and varied, and there should be selected of both drink and diet such articles as appeal to the fancy of the patient. Whenever possible, the patient should live at a certain altitude and should take up an outdoor life, any form of exercise being encouraged that does not produce exhaustion.

In regard to tonics and stimulating foods, it would be well to prescribe cod-liver oil for certain periods and in increasing doses, say from forty to two hundred grammes a day, if the patient digests it. (Many patients have no difficulty at all in swallowing a full glass of cod-liver oil before a meal.) In order to make it more acceptable, the glass should be cooled off by placing it in ice. You have seen in our wards many surprising recoveries from scrofulotuberculosis by means of this powerful treatment.

Where persons do not tolerate cod-liver oil, other forms of fatty food should be prescribed, such as cream, bread and butter, oysters, caviare, and sardines, all of which, with fish and smoked meat, are

valuable constituents of the patient's diet. There may also be given raw meat and meat-juice, which have proved very efficacious in the treatment of tuberculosis. The effect of the meat-juice diet does not consist merely in superalimentation, but appears to be connected with a special antitubercular quality inherent to this preparation.

It would be well to prescribe before meals thirty drops of a mixture of equal parts of tinctures of kola, coca, and cinchona, taken in a small glass of wine or water. Hypodermic injections of sodium cacodylate, one injection of five centigrammes a day for fifteen consecutive days each month, for several months in succession, should be given. The efficacy of this treatment has no longer to be proved; it has stood the severest tests.

Finally, the patient should guard most carefully against exposure to cold; and should remember that years must pass before he can regard himself as safe from any further attack of tuberculosis.

INSECT PESTS OF HUMAN BEINGS.

CLINICAL LECTURE DELIVERED AT THE NEW YORK POLYCLINIC HOSPITAL AND SCHOOL FOR GRADUATES IN MEDICINE.

BY JAMES J. WALSH, M.D., PH.D.,

Lecturer on General Medicine at the New York Polyclinic.

GENTLEMEN,—On a former occasion I showed you some specimens of the larvæ of winged insects that at times find their way into the bodies of human beings, and there cause annoying symptoms.[1] One of them, you may remember, the larva of *Tenebrio obscurus,* found its way into its host probably through the medium of gluten-suppositories used for hemorrhoids. Another was a specimen of the screw-worm. This parasite is not at all uncommon in certain of our Southern States, and as persons are so constantly travelling from one part of the country to the other, physicians wherever situated are likely to see, from time to time, patients suffering from its ravages. As the screw-worm produces its effects within a few days, it is important that the physician who for the first time sees a patient suffering from myiasis should recognize the parasite as soon as possible.

Our recent annexation of one of the West Indian islands, our closer relations with another, the approaching acquisition of the Danish West Indies, and the building of the Isthmian canal in the near future will cause considerably more intercourse than before between our eastern and southern ports and these islands and the countries of Mexico and South America, which will undoubtedly bring with it a more frequent transportation of insect parasites from these more or less tropical regions. Physicians will therefore see the effects caused by such parasites much oftener than before, and it is important that they should be in a position to recognize them.

The practical importance assigned to this subject by masters of medicine may be judged from the fact that the authors of text-

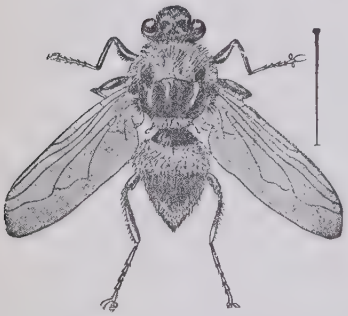
[1] INTERNATIONAL CLINICS, Vol. IV., Eleventh Series, 1902, p. 108.

books of the Practice of Medicine (as, for instance, Professor Osler) now find it advisable to give the subject due consideration in a special chapter, which is only a very recent addition to such works.

BOT-FLIES AND THEIR LARVÆ.

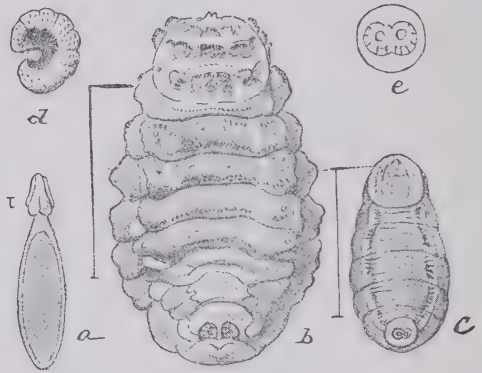
After the screw-worm fly and its larva, the most important winged parasite of man is the so-called bot-fly, or breeze-fly, which belongs to the family of the *Œstridæ;* various species of the fly affect different animals. The lesions produced by the larvæ of the fly are known as the "warbles" in cattle. They are inflammatory, somewhat resembling boils, and occur on the backs of animals as the result of the development of the larvæ from eggs deposited beneath the skin by the insect. The most familiar form of the insect

Fig. 1.



Hypoderma bovis—enlarged (line shows actual length). (After Brauer.) (United States Department of Agriculture, Bulletin No. 5, Division of Entomology.)

Fig. 2.



Hypoderma bovis: a, egg; *b*, full-grown larva, ventral view; *c*, puparium, ventral view; *d*, newly-hatched larva, side view; *e*, anal stigmata of larva—all enlarged (line shows actual length). (After Brauer.) (United States Department of Agriculture, *ut supra.*)

is the ordinary ox bot-fly, or warble bot-fly, the scientific name of which is *Hypoderma bovis.* In its winged stage it looks not unlike a bee; this picture of the insect (Fig. 1), and this of its larva (Fig. 2), are taken from Bulletin No. 5, New Series, of the pamphlets published by the Division of Entomology of the United States Department of Agriculture.

It was questioned for a long while whether this insect ever became a parasite in man, but the observations of trained entomologists have proved conclusively that the larvæ have been found

beneath the human skin. Dr. John Hamilton, who besides being a physician is a trained entomologist, gives the details of a case that came under his personal observation. The patient was a boy of about six years of age, who had been suffering for some months from enlarged glands on one side of the cervical region. There was also a fetid ulceration around the back teeth of the lower jaw of the same side, and this seemed to furnish ample explanation for the existence and persistence of the enlarged glands. Notwithstanding the employment of all the usual methods of treatment, no benefit resulted, and the chronic ulcerative process became more and more extensive. A general septic condition ensued, and a fatal termination was threatened. One day, while making a careful examination of the interior of the mouth, Dr. Hamilton observed a small white object which seemed to move. It was seen just over an ulcer at the root of the tongue, and being extracted proved to be a large grub. Dr. Hamilton, who had frequently seen these under other circumstances, at once recognized it as a full-grown larva of *Hypoderma bovis*. It was of the usual tawny color, about half an inch long when contracted and about one-third of an inch in thickness, and quite lively. In spite of this discovery and continued careful treatment the patient died.

The boy had been on a farm in Illinois during the previous harvest-time. It would seem that in some way the egg of the insect was introduced into his mouth, and lodged perhaps in a crevice between the teeth, or in a bit of loose gum around the socket of a tooth. Here the moisture and warmth proved favorable to its development, and the larva after its hatching found all the conditions favorable to growth between the base of the tongue and the jaw. It came to maturity after about the same interval as do larvæ when deposited in cattle, so that it was nearly six months after its deposition before the grub was discovered and its identity recognized.

It must not be forgotten that particles of matter that get between the teeth, particularly in a farming-country, may prove to be living products that will develop and cause serious damage. The ray fungus or actinomyces micro-organism finds its entrance in just this way into the tissues of cattle from the blades of grain on which it grows; the result is the well-known lumpy jaw so long thought to be a form of malignant disease. A corresponding form of infec-

tion sometimes takes place in human beings, and the actinomyces fungus develops in the human tissues, with the production of a pathological picture not unlike that which occurs in cattle affected by the same disease.

THE HUMAN GADFLY.

Besides the ox bot-fly, which occurs only occasionally in man, there is a genus of this insect which confines its ravages almost wholly to man, the monkey, and the dog; this is known as the *Œstrus hominis*. Considerably more than a century ago, Linnæus seems to have had some knowledge of the fact that this insect sometimes lays its eggs in human beings, where the larvæ develop and not infrequently produce serious consequences. In his "Systema Naturæ" (Volume III., page 583, Curtin's translation, issue of 1802) the following passage occurs: "*Œstrus hominis,* body entirely brown, inhabits South America, deposits its eggs under the skin on the bellies of the natives. The larva if disturbed penetrates deeper and produces an ulcer which frequently becomes fatal to the host of the larva."

Curiously enough, this account published by Linnæus was discredited for many years. Subsequent scientific writers on the subject thought that the species *Œstrus hominis* was a fictitious one, and concluded that the phenomena described by travellers, which had formed the basis for the scientific information published, were the result of faulty observation. Some writers considered Linnæus's mistake due to the fact that the ox bot-fly occasionally deposits its eggs beneath the skin of man and there cause the symptoms described. It was not until about the middle of the nineteenth century that scientific writers began to admit the existence of a special species with a tendency to become a parasite in man. More accurate observation then proved that Linnæus had not been mistaken in his original classification, and that the facts were exactly as he had described them.

A number of articles was written between 1840 and 1860 on the *Œstrus hominis,* or human gadfly. The observations made with regard to this fly form the basis for the numerous legends (some of them going back even to mythological times) of the infestation of man by a special form of winged parasite, which produced upon the human skin ulcerative lesions extremely difficult to heal, at

times causing the death of the sufferer. The old legend of Io shows that the *Œstrus bovis* was well known to the ancients; and though her affliction by the gadfly, while she was in the form of a heifer, is supposed to be symbolic of the stings of conscience for misdeeds with Jupiter, the story is interesting as showing how old the position is with regard to gadflies, and also as giving at least a hint of the possibility of their afflicting human beings.

In more recent years a number of authentic accounts, especially from Cayenne and Mexico, puts the question of the existence of a special gadfly for man beyond all doubt. There is still some dispute as to whether the *Œstrus hominis* may not have preferred originally as its host the monkey, the dog, or other animal living in close intimacy with man, but its present tendency to secure human tissues as the developing place for its larvæ is now very generally recognized.

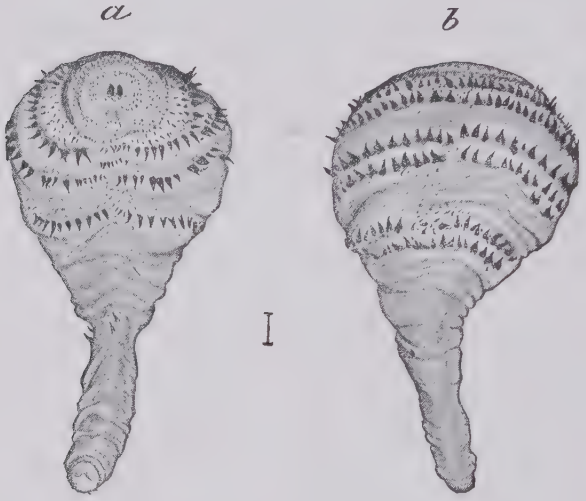
If our commerce with the South American Republics and with Mexico and Central America is to become more extensive than heretofore, as seems almost inevitable, the various names given in these countries to this parasite of man will be of some importance in enabling medical men to recognize it in the stories brought by persons coming from those countries. By knowing the name used in the place from which a patient comes, a physician may be able to gather, even from his own statements, some inkling of the nature of the parasite that is troubling him. He will simply need to find out the name of the gadfly infesting that region.

In Cayenne the species attacking man is called, according to the publications of the Division of Entomology of the United States Department of Agriculture, the *Ver macaque.* In Brazil it is called *Ura* because of the burning sensation produced by the larva of the insect during its growth; in Costa Rica the name is *Torcel;* in Colombia, *Gusano peludo,* or *muche.* Besides these, there is a larva of a somewhat similar gadfly, the *Ver moyocuil,* found particularly in Mexico and Colombia, which lives especially on the dog, but may, it would seem, occasionally affect man. The flies are closely allied to those of the genus *Œstrus hominis,* but the larvæ are long and cylindrical, differing greatly from others of this family in form. Dr. Matas, in an article published in "Insect Life" (Vol. I., page 76), gives an account of a case in which the *Dermatobia noxialis* laid its eggs in the tissues of a man who was taking a bath

in the river during a trip in Spanish Honduras. It seems reasonably certain that the *Dermatobia noxialis* is the same parasite as the *Œstrus hominis* of other writers. Dr. Matas's account shows how easy it is for a visitor to these regions to become the unwitting host of the parasite, and how serious may be the consequences. He gives a series of pictures of the larva as it develops beneath the skin. (Figs. 3 and 4.)

In most accounts of the deposition of eggs in the human being there is no reference to any stinging sensation having been felt at

Fig. 3.



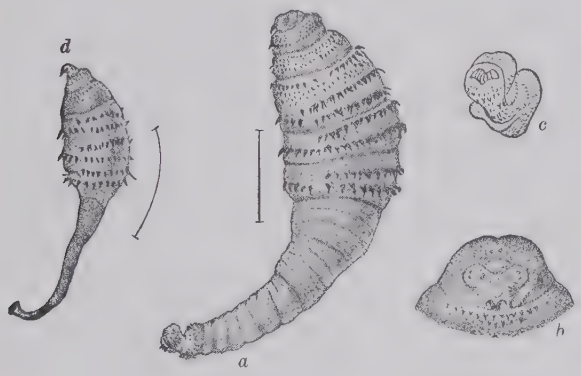
Dermatobia noxialis: larva; *a*, ventral aspect and appearance of cephalic and caudal extremities, also the three rows of spines, single below, and the point where the double dorsal rows end; *b*, dorsal view shows that the three rows of spines single below are double above—greatly enlarged (line shows actual length). (From Insect Life.)

the moment when the oviposition occurred, and victims of the parasite have practically never been aware when the eggs were laid. The only localizing symptom has been the subsequent development of the tumors. Under these circumstances it is easy to understand how there should be doubt as to the form of insect that lays the eggs, and how several names might easily be given to the same parasite. This seems to be actually the case, and the experts of the United States Division of Entomology are convinced of the identity of the dermatobia and œstrus forms, though the stories of their occurrence are obscured by the differences in the terminology em-

ployed. In some cases, however, there is a sensation of pain at the moment the eggs are deposited under the skin.

Usually the incubation period is not very long, and in a week or ten days some symptoms of the growth of the larva are recognized. The area above the location of the larval parasite becomes red and inflamed, and a burning sensation with some boring pain is produced, not unlike that felt when an especially tense boil or furuncle is developing. The supposed boil, however, does not come to a

FIG. 4.



a, Brauer's figure of entire Dermatobia larva, supposed to be closely allied to specimens shown in Fig. 3; *b*, cephalic extremity; *c*, caudal extremity of same specimen; *d*, Dermatobia larva figured by Coquerel, and closely related to, if not identical with, preceding, only seen under lower power and perhaps in earlier development—enlarged (line shows actual length). (From Insect Life.)

head rapidly; the pain increases in severity, and the inflammatory area becomes more wide-spread. In all the cases that have been reported, it is usually at this stage, before it reaches an advanced development, that the parasite is extracted, and consequently nothing is known of its possible pupation-period within human tissues.

The extraction of the parasite is the only treatment necessary, and the lesion usually proceeds to heal without delay or incident, if there has been no secondary infection. If secondary infection has taken place, the abscess, owing to the rather intense irritation caused by the parasite, may extend somewhat deeply into the tissues and will have to be drained. In the countries where the parasite is commonest, the application of tobacco-ashes to the lesion has been suggested, to break down the skin above the parasite and thus facilitate its expulsion. It does not seem probable, however, that

this remedy has any real effect, and its use is only an expectant method of treatment; free incision is the true surgical indication and should not be delayed.

THE HORSE BOT-FLY.

A very common form of bot-fly or gadfly is that which affects horses. Its scientific name is the *Gastrophilus equi,* and the familiar name of the affection which it causes in horses, well known to farmers and stockmen and veterinarians for almost as long as the memory of man runneth, is "bots." These insects do not, it is thought, affect human beings; though there has been some doubt of this, because of certain disorders of nutrition, with tendency to emaciation, which sometimes occur among hostlers and others engaged in the care of horses suffering from the disease. For most physicians, especially in the country, the horse is so important an animal that some slight knowledge of the affection caused by the parasite, and of its method of distribution, are of practical interest, even apart from the possibility of its affecting man himself.

The fly lays its eggs on the hair of horses, especially on the breast and the anterior parts, which are most liable to be licked by the tongue; from here, either in the egg or in the larval state, the insects are conveyed by the tongue into the stomach, and there attach themselves and draw their nourishment from the stomach-wall of the animal. The most curious thing about the insect is the instinct that guides it in laying its eggs on the parts of the animal where the tongue will eventually convey at least some of them into the mouth, and thus enable them to reach the parts of the body best calculated to provide abundant nourishment for the growing larvæ in their early stages. It has been found by experiment that the larvæ are not able to free themselves from the egg by their own exertions, but that external means are required to break the egg-envelope. This force is furnished by the horse's tongue, which exercises just the proper amount of pressure. Then the moisture of the tongue causes it to pick up the freed larvæ. The newly hatched larva is, according to the Division of Entomology of the United States Department of Agriculture, a slender, worm-like creature so transparent that the internal organs are plainly visible. It grows rapidly at first, its food consisting very probably of the mucous secretions of the mouth and œsophagus. As soon as it

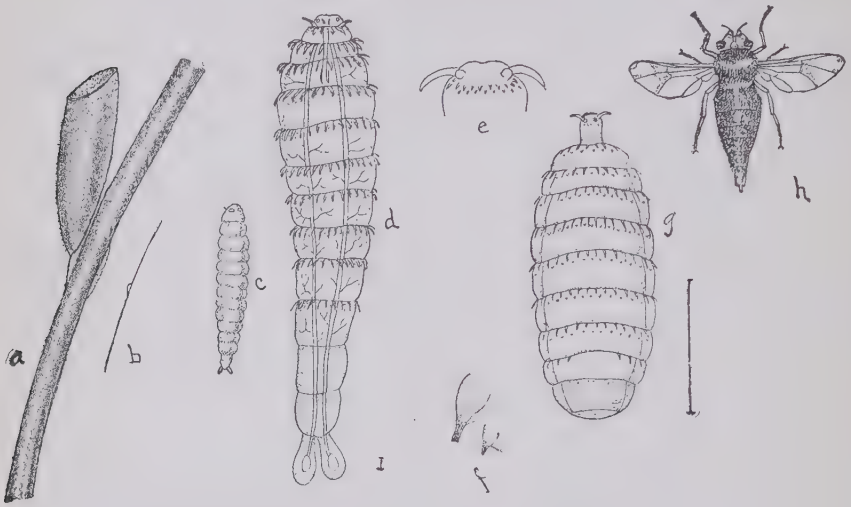
reaches the stomach, it fixes itself to the walls by means of the hooks situated close to its mouth, and soon undergoes considerable change in form and appearance. When full-grown the larva is three-fourths of an inch to one inch in length. The larvæ may occur in large clusters upon the stomach-walls, especially numerous at the pyloric portion, where they serve to cause retention of the contents of the stomach. The growth of the larvæ occupies the period from the late summer when the eggs are laid, through the winter, until late in the spring. At this time they release their hold, and are carried out with the droppings. Then they burrow into the ground, in which they pass the pupa stage. This lasts from thirty to forty days, when the fly issues to proceed with its proper life-work of producing another generation of bots.

Commonly the flies can be observed in the act of depositing their eggs on the legs or body of a horse at work or in a carriage. The animal usually stamps nervously, champs its bit, and is apt to bite at parts where the fly is at work; in spirited horses great excitement may result. The flies may be captured, though this is not so easy a matter. Where horses are curry-combed regularly, the eggs are removed and the larvæ do not find their way into the animals' stomachs. When colts or horses are in pasture, however, there is no curry-combing, eggs are deposited in hundreds, and so the pest, which very seriously disturbs the nutrition of the animal during the winter time, gains its hold. It is during late July, August, and September that the eggs are laid. Horses should be examined carefully every couple of weeks at this time. Rubbing with dilute carbolic acid or with kerosene, clipping the hair, or shaving the eggs off with a sharp knife or a razor, are the best direct prophylactics. If this is done, there will usually be no danger for the animal, and careful inspection of horses in pasture would lessen the possible dangers to those who are in close attendance on them.

It seems very improbable, however, that there is any serious risk for human beings from these insects, notwithstanding the tradition in farming and horse-pasturing districts that the hostlers are liable to suffer somewhat as the horses do. The collections of the larvæ in the stomach of the horse to which all the symptoms are due have often been seen at post-mortem examinations on the animals; they are easily recognizable, and could not be missed at an autopsy on a human subject if they were present. The tradition

remains, however, and there are many obscure affections among farm-hands as yet unexplained. The fly and its egg and larva at various stages may be seen in Fig 5.

Fig. 5.



Gastrophilus equi: a, egg—enlarged; *b*, egg—natural size; *c*, young larva; *d*, young larva—much enlarged, showing spiny armature; *e*, oral hooks; *f*, body-spines; *g*, full-grown larva—twice natural size; *h*, adult female. (Division of Entomology, Bulletin No. 5.)

THE SAND-FLEA, OR JIGGER-FLEA.

Our closer association with the West Indies is apt to bring us many examples of the lesions caused by this noisome little pest. Nearly a century and a half ago Linnæus described this parasitic insect, under the name of *Pulex penetrans;* scientists now call it *Sarcopsylla penetrans,* and it is popularly known as the jigger, or jigger-flea. In the Spanish-speaking countries it is called chigoe or *chiqué,* and it is probably from this that our word jigger is derived. The flea occurs almost everywhere in tropical America and the regions lying near the tropics, and is especially frequent in the West Indian islands. It was introduced into Africa less than thirty years ago, but has propagated with such rapidity that it is now very common there.

Almost any animal may be affected by the jigger-flea. The male jiggers act only as does the ordinary flea, by biting and drawing blood; they have the power to attract blood to the place bitten, by injecting an irritant substance underneath the skin. The female

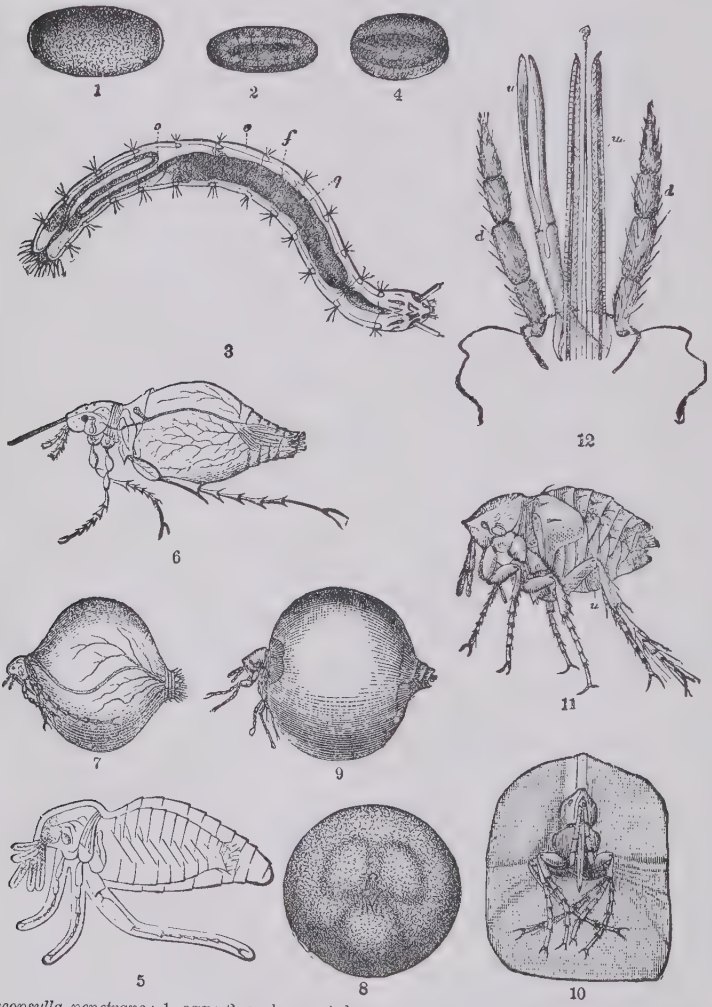
flea, however, after having been impregnated, burrows beneath the skin of its host, a favorite point of entrance being just underneath the toe-nails. Here it produces at first a tender swelling, and then, as the result of inflammatory irritation, a discharging ulcer, which sometimes becomes so serious as to result fatally. The swelling at first is vesicular; if secondary swelling takes place, as is usually the case, a pustular condition develops. The body of the female becomes greatly distended with the development of the eggs, so that it no longer bears the slightest resemblance to the original insect. The Bulletin of the Division of Entomology, already cited, says that the head and legs appear as little appendages upon a round body about the size of a large pea. (Fig. 6, No. 9.) If left undisturbed, the larvæ probably escape in some normal manner from the body of the adult, though it is possible that the development goes on until the egg-sac bursts. Then they go through their further development *in situ,* abstracting nourishment from the tissues of the host.

The remedy most recommended is the removal of the body of the insect as soon as the first tenderness and swelling indicates its presence; experts say that this can be accomplished by the introduction of a knife-point. Great care must be exercised, however, in order to secure the extraction of the insect without rupture of the gestation-sac. The crushing of the body of the mother may result in the premature liberation of the eggs into the tissues, and if they are not promptly expelled from the sore which has already formed, they increase the lesion originally produced by the entrance of the mother, and set up a serious and sometimes even fatal inflammatory process.

The prophylaxis of the affection caused by the insect is not easy. So many animals may become the hosts of this parasite that it is extremely difficult to eradicate it after it has once gained a foothold. Whenever it infests domestic animals, attention should at once be paid to those that suffer from it, in order to prevent the propagation that otherwise inevitably takes place. Whenever adult insects are discovered they should be destroyed. Pyrethrum-powder is of considerable service in preventing the development of larvæ, if dusted freely upon places where they are coming to active life. As a matter of fact, however, such methods have never so far succeeded in eradicating the insect from any place where it has once secured

a foothold. The jigger infests especially the feet, but at times finds its way directly beneath the skin and burrows for some distance; this is particularly apt to occur in human tissues. The use of essen-

FIG. 6.



Sarcopsylla penetrans: 1, egg; 2, embryo; 3, larva; 4, cocoon; 5, pupa; 6, fecundated female; 7, the same on the third day from its entrance under the skin of its human host; 8, the same after several days' residence in the skin of its host; 9, fully-grown female—magnified four times; 10, head of same still more enlarged; 11, female before entering skin; 12, mouth parts much enlarged; *m*, mandibles; *d*, maxillary palpi; *u*, labium. (After Karsten and Guyon.)

tial oils on the feet will, it is said, prevent its attacks on human beings; it may attack other parts of the body, however, and when

a person is bathing in an infested region, it may be necessary to anoint the whole body with the oil.

THE HOUSE-FLEA.

This is the best known species of flea, and has been a familiar pest of mankind for probably several thousand years; it was described by Linnæus more than a century and a half ago. It is very widely distributed over the globe, and, except at the extreme north and south, there is scarcely a place where it may not exist if the proper conditions as to food are present to encourage its multiplication. In the warmer countries it often becomes a serious pest in houses, notwithstanding the most scrupulous attention directed against the accumulation of dust or organic matter, and the most careful domestic cleanliness. It has its favorite location in houses, and finds a lurking-place especially in bed-clothing, or in other clothing stored in dark rooms or closets. These insects more usually attack people at night, but in certain countries they are very common in the dust on the road-sides and readily find their way into the stockings near the ankles, proceeding to draw blood as soon as they become located. They are extremely ravenous, considering their size, and one of them may bite several times in obtaining its due supply of blood.

The breeding-place of the insect is said to be the dust that collects in the corners of rooms and beneath furniture and the like. The eggs are deposited in these accumulations of dust, and the larvæ are said to feed upon the particles of organic matter which may be found in such localities. Each female deposits from eight to twelve whitish, ovoid eggs, about seven millimetres in length by four millimetres in thickness. In summer the larvæ issue from the eggs in from four to six days, and become pupæ about eleven days later. The pupa-stage lasts about twelve days, and then the adult insect is ready to begin its blood-thirsty career. The complete development ordinarily takes about four weeks, in summer, while in winter (according to the Entomological Bureau of our Department of Agriculture) the period is extended to about six weeks. The insect will, of course, develop only in a room which is constantly warmed.

The question of prophylaxis is reasonably important because of the annoyance caused by the insects; there is, besides, as we have learned to realize of late years, a certain amount of danger of the

insect conveying the germs of disease and inoculating them at the point bitten. There has been as yet no specific infection connected with the flea, though there are some good authorities in warmer countries who think that erysipelas of obscure origin, the so-called idiopathic erysipelas, may occasionally be due to the bite of this insect.

A very remarkable fact with regard to the flea and the annoyance produced by its bite is that certain individuals suffer very little, while others are intensely irritated; there may even be a febrile period, with feelings of malaise and some constitutional disturbance, as the result of a series of flea-bites. This has been frequently noted, and appears to be established beyond all doubt. It seems clear too that, as the result of a series of bites, most people acquire a certain amount of immunity from the effects of the poison injected by the insect for the purpose of producing an increased flow of blood to the bitten spot. The exact character of the poison is not definitely known, but it is said to be an acid, which gives the best indication for the treatment of the irritation it causes. The treatment of the flea-bite consists in the application of dilute ammonia, which has sufficient alkalinity to neutralize the acid toxin injected by the flea, and sufficient penetrating power to go to meet it beneath the skin. Besides this, such anæsthetic drugs as cocaine or carbolic acid are useful; touching a flea-bite with a fifty per cent. solution of carbolic acid will usually stop the annoying itching almost at once.

With regard to the cat-and-dog flea (Figs. 7 and 8), which sometimes becomes a pest for human beings also, it must not be forgotten that the insects pass through one stage of their existence apart from the animal. It has been found exceedingly difficult to raise these insects in captivity for study, but Dr. L. O. Howard, the entomologist of the Department of Agriculture, says that the larvæ do not develop successfully in situations where they are likely to be disturbed. In his opinion, the use of carpets and straw matting favors the development of the larvæ, since in the very young stage they can penetrate the interstices of either of these sorts of floor-covering, and find secure abode in some crack, where they are not likely to be disturbed.

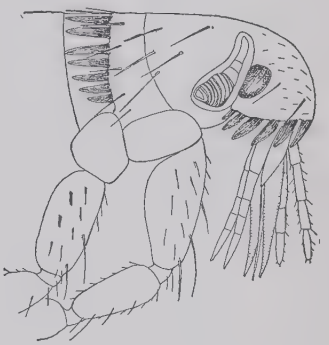
It is comparatively easy to destroy the insect in its early stages, if its presence is detected, but adult fleas are so active and hardy

that they resist successfully any but the most rigorous measures persevered in for a long time. As a rule, the liberal use of benzine over the floor of a house, even when covered with a matting, will suffice to rid it of fleas; certain of the pyrethrum-powders will also probably effect the same purpose. Neither of these remedies, however, is absolutely sure to succeed, and it may be necessary to take up the floor-covering and scrub the floor thoroughly with hot soapsuds in order to secure entire relief. It must be borne in mind, of course, that if benzine is used as plentifully as may be necessary

FIG. 7.

Pulex irritans: head and pronotum, showing form, and absence of combs.

FIG. 8.



Pulex serraticeps: front part of body, showing combs on head and pronotum.

Fig. 7 represents the anterior segments of the ordinary house-flea. Fig. 8 represents the corresponding parts of the cat- and dog-flea. Both are very much enlarged, to bring out the distinguishing characteristics of the two insects. (From Insects Affecting Domestic Animals, issued by the Division of Entomology of the United States Department of Agriculture.)

to give any hope of success, no lights or fires may be allowed in the house for several hours afterwards.

Almost any house where a pet dog or cat is kept may become annoyingly infested with fleas, if the proper conditions of moisture exist, and the larvæ are undisturbed in their development. Pet animals should be provided with a rug to sleep on, which should be frequently shaken and brushed outside of the house. Whenever the rug is moved in the house, or any dust from it is shaken to the floor, the dust should be swept up and burned. Not all the flea-eggs on an infested animal will drop off on to the rug, however, and some of them may develop successfully on the animal's fur. Pyrethrum-powder should occasionally be rubbed into the hair of an animal infested with fleas. If carefully done this will cause

the fleas to drop off either dead or in a lethargic condition; they may then be swept up and burned.

In closing up houses for the summer, it is important to remember that the conditions within the house may be eminently favorable for the development of fleas. Observations made in our climate show that a new generation of fleas comes to maturity about every fortnight, so that if a house which is shut up for the three summer months has but a few fleas at the beginning of the summer, it may easily be infested with them at the end of the summer, as there is usually organic material enough left around the house to foster them. Before shutting up houses, then, benzine should be used plentifully on floors that have been covered by matting, or in rooms where an animal—cat or dog or the like—has been for some time. This precaution is especially necessary if any signs of fleas have been noted for some weeks before the summer outing.

Neurology

BILATERAL CEREBRAL THROMBOSIS DUE TO ATHEROMATOUS CHANGES IN THE ARTERIES.

CLINICAL LECTURE DELIVERED AT THE ARAPAHOE COUNTY HOSPITAL.

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GENTLEMEN,—To-day I will ask your attention to the patient whose history we studied last week and whom I examined before you. Before considering the important points in the case we will hear the history and examination read. The patient, J. D., is fifty years of age, and is a butcher; he was born in Virginia, but has lived in Colorado for the last fifteen years.

Family History.—His father died of apoplexy, and his mother of pulmonary tuberculosis. The rest of the family history is negative. There is no history of cancer in the family.

Previous History.—When a boy he had measles, from which he made a perfect recovery. He has had two attacks of gonorrhœa, the last one in 1879, after which he enjoyed good health until his present illness began. He has been addicted to alcoholic stimulants since he was eighteen years of age, averaging three pints of whiskey a day. He denies syphilis, but his statements are unreliable on account of his mental condition.

Present Illness.—This began one year and a half ago, when he experienced numb and tingling sensations in the left side of the body, and shortly afterwards it was noticed that he dragged the left foot in walking and used the left arm awkwardly. This difficulty in using the left side of the body increased, but the portion was never totally paralyzed. Shortly after the left side was affected he experienced numb and tingling sensations on the right side of the tongue, face, right arm and leg, and also suffered from vertigo. These sensations would last for one-half hour, then disappear and recur after intervals varying from weeks to months. In October,

1901, on reaching home after driving, he was unable to descend from the wagon on account of inability to move the legs, and at this time noticed weakness of both arms as well as difficulty in speech. The following day he was removed to the hospital, and on admission it was found that he was paralyzed in the four extremities, had difficulty in speech, incontinence of urine and fæces, without loss of consciousness.

Examination.—With the dynamometer the right hand gave 50 and the left 42. He was unable to take a few steps without assistance, on account of the marked ataxia and the spastic condition of the muscles of the lower extremities; the latter increased as the muscles were put in use, causing the patient to drag the tip of his shoe. This spasticity was greatest in the right leg and arm, but was present in all four extremities. On attempting to cross the knees he raised the leg in an awkward manner, and to accomplish his object fully he assisted with his hands. There was slight ataxia in the trunk muscles.

Reflexes.—The right knee-jerk was decidedly increased and was increased on reinforcement; the left was increased, but not so much as the right. Ankle clonus was absent on account of the foot being held in extreme dorsal flexion by the spasm; but on relieving the spasm a slight clonus was produced which was continuous in both extremities. The deep reflexes of the right and left forearm were decidedly increased. Those of the right biceps, triceps, deltoid, and pectoralis major were decidedly increased, but not so much as the right. The masseter reflex was absent. The right and left plantar reflexes were slightly increased and the Babinski sign was present. The right and felt cremasteric reflexes were increased, and the lower abdominal right and left, as well as the epigastric, were slightly increased. The right and left tendo Achillis were increased, and on tapping the tendon a clonus was produced.

Sensation.—In the test of the tactile sense hyperæsthesia was found to prevail throughout the entire body. Pressure, joint, posture, localization, and temperature senses were present. Hyperalgesia was everywhere present. The tongue was protruded slightly beyond the teeth, although it was in the median line. There was apparent weakness of the muscles on both sides of the lower portion of the face to voluntary and emotional movements, the latter being more marked than the former. The muscular action of the upper

portion of the face was good. The muscular power in the right and left arm and leg was fairly good for extension, but weak for flexion. In the expansion of the chest the forced expiration gave thirty-four inches, and the inspiration thirty-five and one-half inches. The sense of smell was present and equal. On testing the hearing with a watch, the right and left each measured $\frac{24}{36}$. The tuning-fork was heard best in the closed ear. The perception of taste was present and equal. The pupils of the eyes were equal and responded to light and accommodation. All external ocular movements were good, and the fundi and field of vision were normal. On testing speech it was found that the consonants were slurred, but not to the degree which is found in paretic dementia. During the examination the patient became very emotional, and his memory for recent and past events was very poor, particularly for the former.

The important symptoms to be borne in mind in the case are the occurrence of dizziness and vertigo previous to the appearance of numb and tingling sensations, and weakness of the left side of the body one and a half years ago, the same side subsequently becoming spastic. Later he suffered from numb and tingling sensations on the right side of the face, tongue, arm, and leg, which occurred periodically, and in October, 1901, that side became paralyzed. On admission to the hospital all four extremities were affected, and there was incontinence of urine. The patient could not speak distinctly, although he was conscious of his surroundings.

In making a differential diagnosis of this case we will refer to the various portions of the nervous system in a systematic manner and study the diseases common to each. The only disease of the peripheral nerves which we have to consider is multiple neuritis. The patient was addicted to the use of alcoholic stimulants, and in cases due to this etiological factor there is occasionally incontinence of urine, with increased knee-jerks and other reflexes. The symptoms which would exclude this disease are the involvement of the face, arm, and leg on the left side, and in the course of a month the other side suddenly becoming affected in the same manner. In multiple neuritis the symptoms come on in a symmetrical manner, while in this case they were asymmetrical from the beginning, without any involvement of the sensory phenomena

which is so characteristic of peripheral neuritis. The inability to protrude the tongue, involvement of the trunk muscles, incontinence of urine and fæces, without any disturbance in the electrical reactions or atrophy of the muscles, would exclude multiple neuritis, and disturbance in speech is rarely seen in an affection of the nerves.

We will now consider several diseases of the spinal cord in an effort to account for the symptoms which this case presents, and I will first call your attention to myelitis. We can exclude myelitis here on account of absence of any marked disturbances of tactile, temperature, localization, pain, muscular, or posture sense; although there has been incontinence of urine, it has not been as persistent as it would have been if the lumbar region of the cord were affected. The presence of increased myotatic irritability in the muscles, which at the beginning were paralyzed, and the incontinence of urine, which never occurs in poliomyelitis unless it be complicated by some other disease, would enable us to exclude the latter. The presence of electric irritability to the faradic current and absence of muscular wasting would assist in arriving at a negative view in regard to a lesion of the anterior horns. We can exclude all other diseases of the spinal cord, both of an inflammatory and of a degenerative nature, as the symptoms have been characteristic of a brain lesion throughout the course of the disease. If this be true, what are the lesions to be taken into consideration? Is the patient suffering from a unilateral or a bilateral lesion? One hemisphere of the brain controls the muscular movements concerned in the higher or more differentiated character on the opposite side of the body, also to a slight degree the muscles which act together, such as the intercostals, ocular, and masseter; now, if there were a motor-paralyzing lesion of one side of the brain, the unilaterally acting muscles would be affected, with partial involvement of those of a bilateral nature. The points in this case which would prove that both cerebral hemispheres are affected are the involvement of the four extremities and paresis of the lower portion of the face on both sides with inability to protrude the tongue beyond the teeth. In bilateral lesions of the brain, particularly if sudden in onset, one would naturally think that the medulla or pons would be implicated. If the lesions were in the medulla, there would be involvement of other cranial nerve-

centres, especially the trifacial and abducens, which are in close proximity to the facial nucleus, also of the glossopharyngeal, pneumogastric, and spinal accessory, which are situated very near to the hypoglossal centre. A lesion in this location so extensive would affect not only the lower portion of the face, but the upper as well, and also the nuclei of the facial nerve. Any lesion producing paralysis of one side of the body and lower portion of the face on the same side places the disturbances above the middle of the pons. An extensive or bilateral lesion occurring above the middle of the pons would necessarily produce paralysis of the trifacial nerve, which is not affected in this case. The motor oculi have escaped in this patient; therefore we can exclude lesions of the crura. Granting, then, that the lesion is in the cerebral hemispheres, it befalls us to find the exact location of the trouble. A sudden occlusion of both middle meningeal arteries producing bilateral symptoms would be accompanied by convulsions, loss of consciousness, coma, and death in a short space of time. The acute diseases of the brain can be excluded by the onset and history of this case.

The chronic cerebral diseases which we have to consider are tumor and abscess. In the former there would be the characteristic headache, vomiting, optic neuritis, and later atrophy, which are absent in our patient. Chronic abscesses need not be considered, as we find no point of suppuration in any portion of the body or any of the typical symptoms of the disease.

The sudden lesions of the brain which we now must consider are hemorrhage, embolism, spasm of the cerebral vessels, and thrombosis. The age of the patient is that at which hemorrhage frequently occurs, but the non-involvement of consciousness, with slight disturbance in body temperature and with the premonitory symptoms which have preceded the paralytic ones, would enable us to eliminate hemorrhagic lesions. Bastian, speaking of spasm of the vessels as one of the causes capable of producing a hemiplegic condition, says, "There probably exists in these cases an altered molecular state in the brain tissue of the affected region, which for a time renders it unfit to discharge its proper functions; although, if we could examine the part at the time, there might be no change visible to the naked eye or even by the aid of the microscope." This spasm is seen in epileptic, emotional, and hysterical hemiplegics, and lasts from a few hours to a week or a

month. In this case we find none of the exciting causes, and the paralysis has existed so long that it is not necessary even to consider spasm of the vessels.

Cerebral embolism is usually due to an endocarditis, a fragment of a valve or of vegetation being carried into the cerebral circulation; or it is sometimes produced by a blood state which causes a thrombosis in some peripheral vessel, and a fragment from the plug may be washed into the circle of Willis. In our patient there is no evidence of a cardiac murmur or syphilitic infection. If these extensive bilateral symptoms were due to an embolus, it necessarily must be situated in the basilar artery, but it would produce bilateral symptoms with profound disturbances in consciousness.

The only sudden cerebral vascular lesion which we have left to consider is thrombosis. Our patient complained of dizziness, as well as numb and tingling sensations in the left side of the body for some months, then suddenly became paralyzed without loss of consciousness. After a number of months the opposite side of the body was affected in a similar manner. The presence of premonitory symptoms occurring on both sides and the paralysis coming on without loss of consciousness are typical of a thrombotic occlusion of a cerebral vessel or vessels.

Having arrived at a diagnosis, it is our duty to determine the cause of the affection. Thrombosis is usually due to syphilitic disease of the arteries, to atheroma, or to a blood stasis. The patient has not given any history or exhibited any signs of syphilitic infection, which is the usual cause before forty years of age, although persons after that age are not exempt from such causation. In this man's case we find no evidence that there is an undue tendency to coagulation of the blood. We have narrowed the cause down to atheroma, and the age of our patient is in favor of this; there are similar changes exhibited in other vessels of the body. The changes which take place in the blood-vessel walls are those of a fatty degeneration which becomes calcified, causing a diminution in the caliber of the vessel and favoring coagulation in the diseased areas. In the apoplectic state it is common to have the sphincters affected, causing involuntary passage of the contents of the bladder and bowel; when consciousness returns, the sphincters reach their normal state, except in cases of ordinary

hemiplegia associated with marked mental dulness or in double hemiplegia, where incontinence of urine may persist for years. Sensory symptoms are sometimes met with in hemiplegic cases, and those present in our patient are due to an irritation of the sensory fibres in the posterior third of the internal capsule, causing hyperæsthesia and hyperalgesia throughout the body. We have seen that the small arteries which supply the great ganglia at the base of the brain are terminal and are involved in this case, producing necrotic softening in the areas which they nourish and consequently a degeneration of the pyramidal tracts; this would account for the spastic condition of the muscles and increase of the reflexes throughout the body.

As to the prognosis in this case we will be guided by the history and certain symptoms presented by our patient. The duration of the case would allow us to pass the question as to the immediate danger to life, while, all things being equal, the prognosis is always worse in bilateral than it is in unilateral thrombosis. The presence of decided increase in myotatic irritability which is shown by exaggeration of all the reflexes and the contractures of muscles are positive indications of degeneration of the lateral columns of the spinal cord, which destroys all chance of a recovery. The marked emotional state associated with loss of memory is characteristic of cerebral softening and makes the prognosis more gloomy.

The treatment of the case will consist of a good diet and a certain amount of exercise, care being taken to avoid fatigue. Iron, arsenic, and strychnine should be given, along with ten grains of potassium iodide after each meal. The bladder must be carefully watched, and, if cystitis be detected, should be washed out daily. The remainder of the treatment is on an expectant plan, with an effort to make the patient as comfortable as possible.

TRAUMATIC EPILEPSY; ACUTE MANIA FOLLOWING INJURY; SYRINGOMYELIA; CEREBRAL HEMORRHAGE.

CLINICAL LECTURE DELIVERED AT THE COOK COUNTY HOSPITAL.

BY DANIEL R. BROWER, M.D., LL.D.,

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TRAUMATIC EPILEPSY.

GENTLEMEN,—This patient two years ago began to have attacks of unconsciousness, coming on suddenly and lasting from one to thirty seconds, during which, as there was no premonitory warning, he would frequently fall down and injure himself. He never frothed at the mouth. The attacks sometimes occur from ten to twenty times daily, although occasionally there may be an interval of two weeks between them. Four years ago he was slugged over the occipital region and rendered unconscious, and this severe traumatism seems to have developed these peculiar convulsive seizures. For the last two years he has noticed an impediment in his gait, worse at night; he has no pain in his legs or elsewhere. He is obliged to urinate two or three times nightly; he has lost fifty pounds in weight. There seems to be no patellar-tendon reflex; the pupils are unequal, the left being somewhat smaller than the right. There is a little uncertainty about coördinating with the left arm. There is ataxia of station, and when walking he broadens his base of movement. He has had both gonorrhœa and syphilis. We have here a history of an injury, epileptiform attacks, and symptoms of locomotor ataxia. The attacks are not truly epileptic, as he never froths at the mouth nor bites his tongue, but he does lose consciousness; there is no convulsive movement of the legs, the movements being only in the arm, and he falls backward. This is not, therefore, a typical epilepsy, but an epileptiform consequence of the traumatism.

ACUTE MANIA FOLLOWING INJURY.

This man is not hard of hearing, but always answers in an elevated tone of voice. After falling from a second-story window, he was brought to the hospital in a comatose condition, and could not be aroused by stimulation. The pupils vary in size from contraction to wide dilatation, the left pupil always being the smaller. Respiration is stertorous, and there is slight frothing at the mouth. The pulse is slow and weak. Patellar-tendon reflexes are normal. Upon entrance his right arm was paralyzed, alcoholic breath was detected, and there was no evidence of fracture or depression of the skull. Over the outer angle of the right eye there was a wound one inch long, which bled slightly. Later on, crepitus was found in the right arm due to a fractured radius, accounting for the want of movement in the arm. The wound over the eye was enlarged, in order to make sure that there was no fracture of the skull; it was then closed by suture. A few days afterwards his condition was practically unchanged. He was able to move the right arm slightly after pinching. He became very violent, requiring strapping in bed. Later he became less violent, and this condition remained unchanged for several days. Up to this time he had scarcely spoken, but he could then be aroused sufficiently to say a few words. Thirteen days later the condition had not changed appreciably, but he appeared to be deaf at times, and at other times his hearing was exceedingly acute. I think that he will answer or not, just as he feels inclined. His mental condition also began to improve, and he now knows his friends and reads the paper. Owing to his restlessness, it is impossible to put a plaster cast on his arm. There is no paralysis. The patient is wasting rapidly; he was said to be a very heavy man when he came here. The tongue is protruded slightly to one side. He has been in the hospital now for about seven weeks and his mental condition has remained stationary for quite a while; the violence has subsided, but at times he shows it a trifle. It is a case of acute mania following traumatism.

SYRINGOMYELIA.

Note the deformity of the shoulders and the wasting of the muscles of the left shoulder in the case which I now present to you. Most of the muscles of the left arm are atrophied; the right shoulder droops very much, and there is a slight atrophy of the right

shoulder, not as marked, however, as in the left. This patient was an iron-moulder and consequently must have had great muscular development in order to do his work. The hands are very œdematous, but this œdema is not acromegaly. Notice the little tremor of the muscles, such as I showed you in progressive muscular atrophy,—little tremors and fibrillar twitching of the muscles. There is a similar deformity of the feet, and they are remarkably flat, the arch having completely disappeared.

There is marked scoliosis. Testing his sensibility with the fine brush part of my hammer, we find sensation to be normal over the back, as far as tactile sense is concerned. I stick him in the back with a needle, and he feels the prick; I then introduce the needle for almost half its length, but he does not know that it is there; he felt it touch him, but all sense of pain is lost. Here is an area where there is loss of the touch-sense as well as loss of pain-sense. In the other area there is no pain-sense, but tactile sensation is perfect. Upon testing him for heat and cold, you find he cannot tell the difference, indicating that there is also a loss of temperature-sense. There are numerous places all over the body of this patient in which the sensations are dissociated. Red spots like these where I inserted the needle always follow a pin or needle prick.

Upon testing the chest and abdomen for sensation, we find an area where there is absolutely no pain-sense, although he feels the slightest touch. He cannot tell the difference between heat and cold. We have here the same condition as on the back; the tactile sensation is perfect, while pain and thermic sense are lost.

This man is forty-one years of age, unmarried. The only important circumstance in his previous history is that he had smallpox at the age of seven and has had a gonorrhœal infection. He may also have had a syphilitic infection, although he is not certain. The knee-reflex and the cutaneous reflexes are all exaggerated. Wherever you touch a muscle you get a prompt response. He first noticed some loss of power in the right hand and arm about eleven years ago. This soon extended to the left arm, and about the same time a spastic condition of the legs developed, which has progressed slowly, together with atrophy of the upper-arm muscles. During the course of his work he was exposed to marked variations in temperature. The striking symptoms in this case are the progressive muscular atrophy in

particular territories, not in the places in which you usually find it, in the right hand, arm, index-finger, thumb, and little finger, but a progressive muscular atrophy beginning about the shoulders; also dissociation of sensations, œdema of the extremities, progressive paresis, exaggerated deep and superficial reflexes, and scoliosis.

This is a case of syringomyelia, which means that there are cavities in the spinal cord. These cavities may be of different sizes and in different localities; some show degeneration of the lateral territories, others of the posterior territories, and still others in the anterior horns. They occur normally in the spinal cord in early fœtal life, and gradually disappear during the period of development and growth, the last remnant of them being the central canal of the spinal cord. Occasionally this developmental process does not go on normally, and these cavities remain to a greater or less extent. The acceptance of this explanation does not seem reasonable in this case, because the defects did not come on until late in life. This man was robust up to eleven years ago and able to do work requiring a tremendous muscular capacity in both the legs and the arms. It is not reasonable to suppose that the congenital condition of the cord existed all through his life; on the contrary, it is more probable that some infectious condition set up this degenerative process. Was it the smallpox, or the gonorrhœa, or a possible syphilitic infection? We do not fully realize that gonorrhœa exerts a most destructive effect on the nervous system. Some infectious process must have invaded the spinal cord, producing these cavities. The interesting feature is that the outlines of these cavities are not the same for all segments of the cord. His difficulty began in the right arm. Evidently the right anterior horn was first involved, causing a progressive loss of power, but without any pain; that indicates that there was some destructive lesion in the anterior horn segment. From there it travelled into the other arm and then the legs, then producing these irregular areas, where there is this dissociation of sensation. The extent of these cavities varies in the different segments.

This degeneration is found in all cases that have been examined *post mortem*, and there are now over a hundred of these on record. There is a very slow progressive degeneration, which in this case began eleven years ago. Whether we have any drugs which will stop that degeneration is doubtful. There are only two which

are entitled to any consideration,—namely, the nitrate of silver and arsenic. I administer these drugs alternately for a month at a time, in connection with such other general tonics as may be indicated. In this manner it may be possible to diminish the rate of progression and make him comfortable, but not to cure him.

A very ingenious exposition of syringomyelia has recently been suggested. The theory was advanced that it is a remnant of leprosy. This man is a Swede. There is a good deal of leprosy in Norway and Sweden, and there was a time when it was very prevalent throughout the whole of Europe. During a recent trip in the Sandwich Islands I spent some time studying leprosy. I saw very many feet and hands of lepers which were the exact prototypes of this case. This patient, in fact, looks enough like the lepers I saw to be one of them. There are the same peculiar sensory disturbances in leprosy, though not so marked; and the disorders of nutrition, more marked in some cases than in this one, are also found in leprosy. The loss of finger-nails, together with ulcerations of the toes and finger-tips, accompanies this condition as well as leprosy.

CEREBRAL HEMORRHAGE.

This boy is seven years of age, is very bright, and has a marvellous memory. When eleven months old, while sitting in a child's high chair, he threw himself backward and struck the floor with great violence. He cried considerably after the accident. The nurse picked him up and took him out in his carriage. After he had been out of doors a short time he had a convulsion, and for two or three days convulsions followed one another in rapid succession. With the disappearance of the convulsions, consciousness returned, and he was found to be paralyzed on the right side. He does not raise the toes of the right foot and there is some remaining paralysis of the right leg. The legs are of about the same size; there is no wasting of the muscles. The arms are apparently of equal size. When he walks there is a pronounced dragging of the toes of the right foot, but not of the left. With the left hand he easily throws the dynamometer around to 40 of the outer circle, while with the right he has difficulty in throwing it to 30. In the second attempt he throws it to 50 of the outer circle with the left hand, while with the right the result is the same as in the first attempt. So the relation of his two hands is as 30 to 50, or as 3 to 5. At the time

of the fall something occurred to produce the convulsion, and it has been followed by paralysis. As there is no wasting of the muscles, the convulsions must have been cerebral. I have no doubt that the case is one of cerebral hemorrhage. Where was the hemorrhage? That is an interesting point to determine. For four or five years he continued to have convulsions, with loss of consciousness, but they were limited to the right side. The left side is not paralyzed. The convulsions that he had immediately after the fall were also one-sided, so that it seems reasonable to locate the hemorrhagic focus in the motor territory in the opposite side, or to regard the hemorrhage as meningeal, and not as having occurred where cerebral hemorrhage more frequently is found,—in the neighborhood of the corpora striata ophthalmica of the basal ganglia. Owing to the fact that there was a source of irritation on the opposite side of the brain sufficient to produce convulsive movements on the paralyzed side, the probability is that there was a cortical hemorrhage either of the vessels of the dura mater or of the vessels of the pia mater.

Another interesting feature about the case is that two years ago the convulsive seizures disappeared. You know that epileptiform attacks are of two kinds,—the *grand mal* and the *petit mal*. For some reason or other, two years ago the *grand mal* seizures stopped. At present, once a week, sometimes more frequently, he has fits of *petit mal*. This is a very unusual course; the reverse is more common: a child will suffer from *petit mal* for a long time, and after a while have attacks of *grand mal*. The seizures are momentary, during which he grinds his teeth for a few seconds, and the loss of consciousness is not sufficiently prolonged to cause him to fall. There is a little twitching of the facial muscles, but the setting of the teeth and the movement of the muscles of the lips quickly disappear. Possibly the hemorrhage took place in the cortical territory when he was eleven months old. At one time there must have been a great deal of irritation in that region, when he had the *grand mal* attacks. Now there is less irritation, and he simply has the *petit mal* seizures.

If my theory of the boy's case is correct, and I think there is a great deal to be said in favor of it, the thing to do is to stimulate absorption so as to remove what remains of the hemorrhagic focus. For that purpose I would give him iodide of potassium, and, in

order to control the explosive efforts of the cells of the brain that produce the *petit mal* attacks, I would give him bromides. A combination of the iodides and bromides would be the treatment indicated in his case. Again, although it is disputed by some, my personal observations lead me to believe that we can send a galvanic current through the brain, and thus bring about changes in the nutrition of the head. I would apply one large electrode over the motor territory and the other large one on the opposite side, passing the current transversely through the brain; and, to stimulate the vasomotor apparatus of the brain, send a current also from the forehead to the nape of the neck, the negative pole being placed over the supposed seat of the lesion and the positive on the opposite side. If the combined treatment does not stop the *petit mal* seizures, I would have a surgeon trephine a portion of the skull over the motor territories, at least for exploratory purposes, as this can be done without any particular injury to the boy. Under the antiseptic surgery of to-day there is less danger in opening the skull than there formerly was in cutting off the first joint of the finger. Exploration of this child's head might result in some permanent good. I regard as encouraging the fact that the *grand mal* seizures have disappeared and attacks of *petit mal* have taken their place; usually as a child grows older the pathological process deepens and the *petit mal* becomes *grand mal.*

Our next patient was born in England sixty-five years ago. He uses alcohol and tobacco. His father died of bronchitis, and he has had several attacks of the same disease. While walking up-stairs yesterday morning he noticed weakness in his legs, which grew worse until he had absolutely no use of his left leg and only partial use of his left arm. The patient's general nutrition is fairly good. At the present time he is unable to move the left leg, but can move the left arm a little. The tongue is protruded in the median line; pupils are equal and respond equally to light. The patellar-tendon reflex is exaggerated on the left side. There are many coarse râles over the entire chest; the heart is feeble; there are no murmurs demonstrable. This man, like the boy, has hemiplegia, but here it occurs in one sixty-five years of age with no history of an accident. The man was addicted to alcohol. Arterial degeneration is noticeable in both the radials and is also well marked in the femorals. What produced the paralysis in this case?

Let us see what is the difference in power on the two sides. With the left hand he throws the indicator of the dynamometer to 45 on the outer circle, while with the right he throws it around to 55. Now, what has produced the paralysis? Hemiplegia from spinal disease is extremely uncommon; so is hemiplegia from degeneration of nerves. A neuritis that would affect the two sides in this way would also be exceedingly rare. Hence I think we can safely say that whatever the lesion is in this man's case it is in the brain. As most cases of hemiplegia follow hemorrhage, we might say that the probabilities here are in favor of hemorrhage simply because it is the usual cause; but we have another reason for saying that this case is one of hemorrhage following the rupture of a blood-vessel, while the other two possible causes are from the plugging of a blood-vessel. In brief, we have to consider cerebral hemorrhage, cerebral embolism, and cerebral thrombosis. Which of the three is probably the lesion here? Plugging of a vessel in the brain by embolism—that is, by a clot brought from a distant point—is unusual in persons as old as this patient, because the embolus generally comes from the heart. A patient with rheumatism has an endocarditis and polypoid deposits upon the valves of the heart; some of these growths, whipped away by the blood stream, almost invariably travel into the left middle cerebral artery. So far as embolism is concerned the location is right; but this man has no valvular disease of the heart and he has reached a time of life when acute articular rheumatism is very uncommon Hence, I think we can safely exclude embolism. How about thrombosis? Apart from bronchitis, his health previous to the attack was good; there was no trouble with the head and no dizziness. Thrombosis is usually caused by endarteritis, and this is frequently syphilitic. As syphilis is in most cases contracted in early life, endarteritis is a condition usually found then. He may have endarteritis from other causes,—from interstitial nephritis, for example,—but urinalysis excludes this. An endarteritis has some such history as this: the interior coat of a blood-vessel becomes roughened, the fibrin from the blood becomes deposited there and shuts up the vessel temporarily; then the patient has a little impairment of function, a sense of numbness or of weakness, that passes away and recurs after a time. He will have a good deal of headache, from the disturbance of the circulation in the brain

that results from a partial occlusion of blood-vessels by the thrombotic process. Thrombosis is rarely confined to a particular territory; many points of the brain are attacked at once. The patient's previous good health and the absence of any well-defined cerebral symptoms prior to this paralytic stroke negative the thrombotic theory; hence the probabilities are that the lesion in the brain which produced the hemiplegia was hemorrhage. There is a very marked degeneration of the blood-vessels; they are like pipe-stems and will break very easily. Degenerated blood-vessels in the brain will break much more rapidly than those in other parts of the body, because the cerebral blood-vessels are not supported by contiguous tissues as blood-vessels generally are. In the brain each blood-vessel, being surrounded by a perivascular space, lacks the support that the brain tissue might give it, so that, when degeneration occurs, to a certain extent hemorrhage usually takes place first in the brain. Considering the man's age, history, and the pipe-stem character of his arteries, the probabilities are altogether in favor of his paralysis being the result of hemorrhage. There is no particular difference in the treatment of acute softening from embolism or thrombosis and destruction of the brain by the hemorrhagic process.

Another interesting feature about this man's case is the method of its onset, as he has what is known as the simple form. He did not lose consciousness. Most of the cases of cerebral hemorrhage come on in the form of apoplectic seizures. If standing, the patient falls, becomes unconscious, and passes into a state of profound coma, from which he cannot be aroused by ordinary methods. The respiration is peculiar: with each expiration the cheeks are puffed out, but after each inspiration they are drawn up, and the flapping in and upon the teeth gives a peculiar quality to the respiration that belongs to apoplexy, whether it be due to hemorrhage, embolism, or thrombosis. The temperature is subnormal during the comatose state; it frequently falls as low as 96½° F. As a rule, during the apoplectic fit there is conjugate deviation of the head and eyes. The head is turned away from the paralyzed side, and it is almost impossible to get it back into the median line. The conjugate deviation of the head and eyes is a very important diagnostic feature. You can usually in the midst of the coma detect a little difference in the muscular tonus of the two

sides. The laity have great confidence in a person who can look into the future; and if you can say positively that a man has apoplexy and will be paralyzed and your prediction proves to be true, your prognosis will give you a great deal more reputation than a correct diagnosis.

Again, as onset may be epileptiform, the man loses consciousness and falls. This indicates a more serious hemorrhage usually.

What shall we do for the patient? If called to see a person in an apoplectic or ordinary seizure, the first thing to do is to make a diagnosis. The coma of apoplexy is very much like that of alcoholic coma. The coma of uræmia is very much the same; so is the coma of diabetes. Hysterical coma is sometimes difficult to differentiate from the other conditions that have been mentioned. Alcoholic coma—the ordinary comatose state that produces respiration something like that of cerebral hemorrhage—is never profound unless there are some complications in the case. Pressure over the supra-orbital foramen in alcoholic coma will cause a man to come to immediately.

The value of this simple diagnostic expedient is forcibly illustrated by the unfortunate experience of an eminent physician of this city, who, on going out to attend obstetrical cases, took a drink of whiskey to fortify himself for the night's work before him. On his way home he fell down in an apoplectic fit, and the good doctor who found him with a strong odor about his breath simply said that he was "dead drunk." It was twenty-four hours before the doctor learned that the distinguished physician was not intoxicated, but paralyzed.

The coma of uræmia is very much like that of cerebral embolism and thrombosis. There is no easy way to differentiate it. If you examine the urine, you may have reason to suppose the case is uræmic coma, but there are one or two points that will help you out. The temperature in the coma of apoplexy is subnormal, and if you find conjugate deviation of the head and subnormal temperature, you know that the difficulty is not uræmia, and that it is not opium poisoning or diabetic coma.

This coma is a condition in which you can do very little for the patient. A purgative should be administered as soon as possible, to relieve the intracranial pressure. In the comatose state you may place on the patient's tongue one or two drops of cro-

ton oil, and if arterial tension is exceedingly high, it is well to draw blood. You may, therefore, apply leeches to the temple, behind the ear, or you can draw blood from the arm. When consciousness returns, if the blood-pressure is high, the administration of aconite is a good thing, using it every few minutes until the high tension of the pulse is diminished. Shut out the light and keep away the friends. If there be retention of urine, catheterize the patient. For the next few days he will suffer from headache and restlessness, which you can best relieve by the administration of moderate doses of the bromides. Later, if there be no inflammatory disturbance, nature undertakes to shut off the area of acute softening from the balance of the territory of the brain by encapsulating or surrounding it with connective tissue. Whether it be an area of acute softening from embolism, thrombosis, or a hemorrhagic focus, in either case it is encapsulated. This process is accompanied by fever, and sometimes by restlessness and delirium. You give a purgative and then the bromide. When you get beyond the inflammatory condition, a week or two after the accident, it is time to think of means to restore power. You may from the beginning of the case resort to moderate massage. Later on, you may use electricity; and, believing that the galvanic current has alterative properties, promotes absorption, stimulates function, and improves nutrition, I should send the galvanic current through the patient's head and exercise the muscles of the paralyzed side by the faradic current. In paralysis from brain disease there is no wasting of the muscles except what comes from disuse, so the faradic current will readily cause response. Then comes the general nutrition of the patient; whatever builds him up benefits him in this particular direction and should be given. Elimination by the kidneys and bowels should be carefully attended to, and tonics, such as iron, quinine, and strychnine, with small doses of iodide of potassium and the mild use of laxatives, constitute the general constitutional treatment indicated in this case. From the beginning the patient must be instructed to exercise on the paralyzed muscles all the will-power that he possesses. In a certain number of cases you will in the course of three or four months find contractions coming on. This man perfectly straightens out his fingers. After a while he will have contractions: the fingers will be drawn in so that he cannot extend them; there will be also more or

less permanent flexion of the elbow. Rigidity first occurs in the hand, and later in the foot. When this comes on, you know that the case is one of secondary lateral sclerosis in consequence of a descending degeneration that began in the hemorrhagic focus or the area of the softening and extended down through the internal capsule, the crus cerebri, the pons, and from the middle into the lateral territories of the spinal cord. You also realize that you have accomplished all that is possible for the restoration of the patient, and it is a good thing to tell him or his friends that there is no use in further treatment, as there will never be any further improvement in power. Of course, they may get tired of you and go to some other doctor, but very soon you will get the benefit of your correct and honest prognostication.

It is remarkable that this man should have so much degeneration of blood-vessels at sixty-five years of age. I believe that the progress of this atheromatous degeneration may be retarded by the line of treatment proposed by Professor Bartholow, which consists in the internal administration of alkalies and alteratives,—the carbonate of ammonium and the iodide of ammonium. They increase the alkalinity of the blood and promote elimination. Along with these may be given malt and the hypophosphites. You must impress upon the patient the importance of keeping himself free from excitement; he must not indulge in alcoholics and the luxuries of the table. He must lead a very quiet and peaceful life, avoiding all causes that tend to increase the blood-pressure of the brain. By so doing something can be done towards preventing the recurrence of hemorrhage in these cases as well as in others where there is such marked degeneration of blood-vessels.

INFANTILE SPINAL PARALYSIS.

CLINICAL LECTURE DELIVERED AT THE KENTUCKY SCHOOL OF MEDICINE HOSPITAL.

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GENTLEMEN,—This is an interesting case of infantile spinal paralysis that we have had under observation in this clinic for a long time. Two years ago last September this child, now about five years old, was taken with what the mother described as a fever; my assistant found the child with a temperature of 102° or 103° F., seemingly in a serious condition; when the mother handled the child it evinced great pain, especially in its lower limbs; the child was unable to stand on its feet, and had to be lifted and turned. The high fever ran a course of four or five days, and after it had subsided the mother noticed that the child was unable to stand. When I first saw the little patient, soon afterwards, I found it completely paralyzed in the lower limbs,—a complete paraplegia. This paraplegia continued for nearly a year, the boy possibly showing some little movement in his legs as the months went by. By the end of a year the mother found that he could pull himself up along the edge of a chair, and the improvement from that time to this has been gradual, but positive, until he is now able to walk into the amphitheatre.

This represents a typical case of infantile spinal paralysis, or what is termed acute anterior poliomyelitis. This is not an uncommon disease in childhood. It occurs ordinarily between the second and the third year, about the time of completion of dentition or of the change from the primitive milk diet to the more mixed diet of later childhood. It is a disease of the anterior cornua of the spinal cord, affecting the multipolar ganglionic cells, which preside over nutrition. The portions of the spinal cord most frequently affected are the cervical or the lumbar region, resulting in paralysis of the legs or arms. Does the morbid process begin in the neuroglia? Does it start in the blood-vessels and extend to the

multipolar cells, or does it affect primarily the ganglionic cells and secondarily the neuroglia? These questions have not been answered, for in the few cases that have died from this disease or early complications, both the neuroglia and the multipolar cells were found involved; and in those cases that have lived for years and then died of other troubles, post-mortem examination has shown localized degeneration. The anterior cornua are more highly developed in the child than any other portions of the spinal cord; they are in a higher state of nutrition, out of proportion to the other regions of the cord; hence their tendency to inflammation in childhood is far greater than it is in the adult.

Etiology.—We find that possibly four-fifths of all cases of infantile paralysis come on between the months of June and September, and that the great majority of this eighty per cent. occur during the latter part of July, August, and the first part of September; they occur generally in the summer months, when the child's vitality is lowered by the intense heat and its nutrition is impaired by gastro-intestinal disorders so frequent at this season, while improper clothing, sudden exposures to cold, and sudden chilling of the surface of the body may be factors in inducing this condition. It is during this period that the clothing of the child is not changed according to season; it will practically have no clothing on during the day, or will be allowed to get very warm, run out in the rain, or be exposed to cold winds, or exposed at night without sufficient clothing, and the surface of the body will become thoroughly chilled. I believe that exposure to cold may be an important factor in these cases. As a result of this combination of unfavorable conditions we find that the most important etiological factor at this period is the part played by intestinal toxæmia, about which a great deal has been written in the last few years. That intestinal toxæmias are capable of producing inflammatory changes in the anterior cornua of the spinal cord is now well established, but just how toxæmia plays this part we are unable to say. A specific organism has not yet been demonstrated.

Symptoms.—These may be divided into the acute, subacute, and chronic forms of anterior poliomyelitis. The usual history of a case is very much like that which we have given you. The child may be ordinarily robust, healthy, and well-nourished; it will possibly be a little fretful and peevish for a day or two, when

it will suddenly be taken sick with fever, the temperature ranging from 101° to 103° F. No evidence of paralysis will develop at first; the fever ordinarily is not very great, and the mother may think it is due to some intestinal condition and give a mild purgative and look after the general condition of the child. Then in two or three days the nurse will find that the child is unable to stand on its feet, or it may be unable to use one or both its upper limbs. She will then send for the physician, as a rule, and he will find evidences of paralysis. Again, this condition may develop without any special prodrome, without any special constitutional symptoms that would lead the mother to suspect that the child was anything except a little peevish; it will go to bed perfectly well, and when the mother attempts to take it up to dress it in the morning she will find that the child is unable to stand, or unable to use one or both arms, the condition having come on suddenly.

As with all of the acute fevers of childhood, acute poliomyelitis may be ushered in with a chill, or instead of a chill there may be a convulsion, the convulsion of childhood taking the place of the initial chill of the infectious fevers in the adult. In the beginning you will usually find that the child complains of soreness and tenderness in the muscles that are paralyzed or in the paralyzed limbs; but this soreness ordinarily disappears in twenty-four to forty-eight hours, or within a short time after the febrile period has passed, and the rule is that when the physician sees the case he finds little or no soreness manifested. The paralysis in the beginning is very frequently a paraplegia, in fact, the lumbar region is the one most frequently affected. A number of cases of hemiplegia are found, because the cervical portion of the cord may be affected as well as the lumbar portion. My experience, however, is that, as a rule, we have the paraplegia continuing for a week, a month, two months, or even a year; after a time, however, certain groups of muscles will begin to regain their normal activity and normal strength; the limbs will clear up, leaving only one muscle or a certain group of muscles paralyzed. It may be the anterior tibial group, it may be the adductors or extensors, it may be the posterior tibial group, or it may be the adductors or extensors of the hand or forearm. The muscles most frequently affected, which are left paralyzed, are the extensors, adductors, and supinators.

While cases of paralysis of the trunk muscles have been reported that are undoubtedly due to anterior poliomyelitis, this condition must be extremely rare; I have seen possibly but one case where paralysis and atrophy of the trunk muscles could be attributed to poliomyelitis. That condition supervenes in pseudo-hypertrophic paralysis, another form of paralysis of childhood, but the trunk muscles are usually unaffected in acute poliomyelitis.

In addition to these symptoms there is paralysis of motion and not of sensation. When the paralysis has become well established in a very short time the muscle or group of muscles begins to shrink, and there is marked atrophy of the muscles or groups of muscles affected. The principal characteristics of the disease are paralysis of the muscle and marked atrophy from nutritive disturbances. The limb will be in a flaccid condition, which distinguishes these cases from the spastic paralysis of cerebral origin. You will find that the temperature of the affected group of muscles is lowered. The circulation is poor and the skin generally shows a mottled appearance. The limb is colorless, almost cyanotic, and there is a difference of one to one and a half degrees in temperature between the affected and unaffected parts. There is a peculiar flail-like motion of this boy's legs as he walks, shown particularly in the left limb, which appears to be perfectly flaccid and loose, there being no spastic paralysis whatever. This child had to learn to walk just as if he were a mere baby, first beginning to crawl; then pulling himself up by the side of a chair, supporting his body or his limbs by this means or with the assistance of his mother; then finally walking as well as he does now.

In addition to paralysis and atrophy of the muscles we sometimes see atrophy of the ligamentous tendons around the joint, which gives us a loose or flail joint, as this boy has at the knee, showing a subluxation of the knee-joint. In this case there is no atrophy of the bones; these have continued to grow though the muscles atrophy, and although there is deformity by paralysis of the muscles, there is no deformity of the bony structures. The bones in the majority of cases continue to grow; on the other hand there are cases with failure of growth of the bony structures as well as atrophy of the muscular elements. The results of this may often be seen in adults who have small, almost infantile, hands, forearms, and lower portions of the leg.

Differential Diagnosis.—Let me state in a few words the differential diagnosis between the cerebral paralyses and the spinal paralyses of childhood, particularly acute anterior poliomyelitis. We find the cerebral palsies generally affecting one side, giving us hemiplegia. It is true that we have this variety of paralysis in poliomyelitis, but the most common form is monoplegia or paraplegia. We have in all cases of cerebral paralysis, whether in children or adults, a certain amount of spastic rigidity of the muscles. In infantile paralysis we have a flaccid condition of the muscles as is seen in the case before us. Then, too, with the spastic rigidity of the muscles you would naturally expect a restrained or spastic gait, in contradistinction to the flaccid gait which is seen in this child. In cerebral paralysis the whole limb, all the muscles, and all groups of muscles of the limb are affected; whereas in poliomyelitis, as far as the ultimate effect is concerned, we find only certain muscles or groups of muscles affected, *i.e.*, the extensors, adductors, and supinators. It may be one muscle or one group; it may be one supinator or it may be all; it may involve one extensor or many of them; there may be a localized degeneration if you choose to call it so. In cerebral paralysis we have contraction of all the muscles affected eventually, especially the flexors and adductors; whereas the only contractions occurring in poliomyelitis are in the flexors of the calf. When the gastrocnemius is involved, there being no paralysis of the flexors, a form of equinus is produced; then if the anterior tibial group is involved, the gastrocnemius not being affected, you have produced another form of equinus. As to spasmodic movements in the two conditions, in cerebral paralysis we have the characteristic athetoid movements, while in poliomyelitis anterior acuta we have no athetosis. In cerebral cases we may have postparalytic chorea, following hemorrhage into the brain or meninges. Epileptic convulsions are not uncommon in cerebral paralysis, whereas in poliomyelitis the only convulsions that occur are in the beginning of the attack, just as we have the initial chill in other conditions. Sensation is unaffected in both diseases.

In cerebral paralysis we have simply an arrest of growth, and no impairment of the nutrition of the part other than the impairment that results from non-use; whereas in poliomyelitis there is a tendency to extreme atrophy, extreme interference with nutrition,

coming on early in the paralyzed limb and extending until the muscles of the limb become simply shreds in some cases, the muscular fibres being almost entirely replaced by fatty fibres, leaving the muscle completely atrophied and practically destroyed.

In cerebral paralysis we have normal electrical reaction, whereas in the paralysis of anterior poliomyelitis we find the reaction of degeneration, which is positive proof of anterior poliomyelitis if it is properly applied; but it is a method of examination that is oftentimes a failure in children if not properly used. The patellar-tendon reflexes are exaggerated in cerebral paralysis, whereas in poliomyelitis, when the anterior tibial group or the quadriceps extensor muscles are involved, you have an absence of the tendon reflexes in the affected limb. In the majority of cases of cerebral paralysis we find the speech is more or less impaired, whereas in poliomyelitis there is no impairment of speech. In cerebral paralysis, as a rule, the intelligence is impaired, whereas in poliomyelitis intelligence is normal.

Treatment.—The treatment in the acute stage resolves itself into absolute rest and quiet. You should endeavor to prevent further intestinal toxæmia, and if fever is pronounced when you see the case, give some of the milder refrigerating remedies such as aconite, liquor ammonii acetatis, ammonium bromide, phenacetin, etc. Keep the affected limb or limbs warm by dry applications, protect from chilling; keep the bowels well open. The salicylates may be used if the soreness and tenderness are marked and persistent. Ergot and belladonna, theoretically, should be of some value. After the acute stage has subsided, gentle massage and heat may be used, along with faradism when response is gotten,—otherwise galvanism.

Chronic changes and contractions should receive proper attention. The subject of tendon transplantation is receiving a great deal of attention from our surgeons. Four methods have been employed: (1) Dividing the tendon of a healthy muscle and suturing its central portion to the tendon of the paralyzed muscle. (2) Dividing the tendon of the paralyzed muscle and attaching its peripheral portion to the tendon of a healthy muscle. (3) Splitting the tendon of a healthy muscle and transplanting only part of the tendon to the tendon of the paralyzed muscle. (4) The periosteal method, in which transplanted tendons are attached directly to the periosteum.

Surgery

REMARKS ON SOME EFFECTS OF FIRE-ARMS AT SHORT RANGE, WITH EXPERIMENTAL ILLUSTRATIONS.

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I DESIRE to draw attention to certain effects and appearances produced by the discharge of fire-arms at short range, especially of pistols.

In the *Boston Medical and Surgical Journal* for October 2, 1884, Dr. D. B. N. Fish, of Amherst, Massachusetts, published a paper on the external appearances of pistol wounds. Dr. Fish served as surgeon to the Twenty-seventh Massachusetts Regiment in our war of 1861, and was afterwards one of the medical examiners for the State of Massachusetts. He had been greatly interested in wounds by fire-arms, had made many experiments on the effects of pistol shots at close range, and had arrived at some remarkable and original results. In the summer of 1893 I spent some time at a sea-shore hotel with Dr. Fish. He then told me of his work, and together we repeated many of the experiments.

The conclusions demonstrated were that in pistol and revolver shots the gases resulting from the explosion did not pass with the ball through the skin, but were expended in great part on the surface and tissues surrounding the opening made by the bullet. A scorching of this circumferential surface (in animal tissues the skin at the wound of entrance) was thus produced, always present in shots at six inches, nearly always in those at from six to twelve inches, with powder blackening to a much greater distance. This blackening is in part due to the smut of the barrel, which after repeated discharges becomes very great, to the smoke, to the heat and flame of burning gases, to partly burned powder, to the ashes

PLATE SHOWING COMPARATIVE EFFECTS OF "BLACK" AND "SMOKELESS" POWDER CARTRIDGES AT SHORT RANGE.

Figs. 1, 2, 3, and 4 are produced by a revolver (.38 inch) charged with ordinary black powder and held in hand. The firing distance is eight inches. The markings were made before the members of the Academy of Surgery of Philadelphia on the evening of May 5, 1902. In Fig. 1 the hammer is up; Fig. 2, to the left; Fig. 3, to the right; Fig. 4, down. Fig. 5, revolver immovably held by fixing in a vice; bullet-hole in centre of brand; black-powder cartridge. Fig. 6, revolver cartridge of "smokeless" powder, fired from hand; hammer up; showing circular powder mark, with little or no "brand"; Fig. 7, same, hammer down. (The illustrations are about one-half natural size.)

of burned powder, and to the partly burned and unburned powder grains driven into the tissues. Dr. Fish had also noticed and drawn attention to the fact that the burned and blackened spot, or "brand," as he termed it, held a constant position to the bullet-hole, being above, below, or to one side according to the position in which the pistol was held at the moment of discharge; so that from the position of the brand it was possible always to tell the position of the pistol fired within certain close ranges. In more than two hundred experiments made by Dr. Fish with seventeen different pistols and revolvers, smooth and rifled, of various calibers, fired with loose ammunition and with cartridges, the result was unvarying. This fixed location of the brand was due to the recoil of the muzzle of the weapon in the direction of its sight. Prevent this recoil, or "kick," by holding the weapon immovably in a fixed vice, and the bullet-hole will appear in the centre of the burned spot, instead of at one side.

Dr. I. N. Hall, of Sterling, Colorado,[1] who reports a case of homicide quoted below, thus explains the recoil or kick. He says that the pistol is usually so held as to be supported by the hand below the line of application of the force generated by the burning powder, and this force tends to cause the weapon to revolve around the point of support. "The gases which impel the bullet are necessarily behind it, and follow the new direction of the barrel after the bullet has left it, and hence strike above, below, or to one side of the bullet-hole. The powder stain may surround the bullet-hole, but the brand follows the rule." The slowness of the explosion would thus seem to determine the position of the brand. In the comparatively instantaneous explosion of the smokeless (nitroglycerin) powder we have the circumferential blackening, but not the brand.

I have said that the position of the weapon fired at close range can be determined. This is the rule as given by Dr. Fish, but I must here remark that it applies only when black powder is used. It does not hold good, as we shall see, when the smokeless or gray powder is employed.

"Whenever this burned or smutted spot is found, either upon the skin, the hair, or the clothing at one side of the wound made by

[1] Boston Medical and Surgical Journal, August 14, 1890.

the bullet, by placing the muzzle of the weapon upon the wound so that the line of the hammer and sight will meet a line drawn from the centre of the spot of burning, or of burning and smutting, you will have the exact position of the weapon when it was fired." The brand will be well marked from a small revolver thus fired at from six to eight inches and from a weapon of large size at from nine to twelve inches.

It must be noted that in the modern hammerless weapon the phenomena of powder mark and brand are identical with those described in the ordinary arm with hammer. The sight on the barrel will serve to indicate the imaginary position of the absent hammer.

The importance of a knowledge of these effects in the consideration of questions of suicide, murder, and accident is evident. In practical illustration I may refer to the case of homicide reported by Dr. Hall,[1] in which the position of the brand was just *below* the bullet wound. The defendant claimed, and the jury after personal experimentation accepted the fact, that the weapon was held with the hammer downward when fired. Several witnesses in corroboration stated that in the scuffle in the dark the pistol was discharged in the defendant's act of striking an overhand blow, at the time the pistol flash was seen, without any effort or attempt to shoot and kill. A verdict of acquittal was rendered.

In the matter of suicide by pistol it must be noted that there are not a few impossible suicidal positions,—*e.g.,* on the back of the head, with hammer and sight forward, or forward and downward; over the heart, hammer downward and inward, and perhaps downward; on the side, hammer and sight backward. (Fish.) At the same time ingenious suicides may be produced in apparently impossible positions and seemingly beyond detection.

I may here allude to the effect of blank cartridges at short range. These are usually thought to be devoid of danger, though I have often observed over a large area the scattering and tattooing from the unburned powder grains, producing a burning or charring effect as great as—indeed, almost greater than—if the cartridges were discharged with a bullet. The burning effect of close pistol shots is shown on cotton and linen stuff by the serpentine creeping of

[1] Paper read before the Colorado Medical Society, June 18, 1890.

the combustion in the direction of the brand. It should be remembered that the odor of burnt hair may persist for a long while.

During the summer of 1901 I repeated with great care, and at length, the experiments of Dr. Fish, obtaining always the results he has faithfully described. His experiments and his results were based upon the explosion of black powder, either loose or in cartridges. He did not, I believe, enter into the study of the effects of smokeless powder. At the time of his death, six or seven years ago, its use in cartridges had not become common. In my own experiments I employed not only the ordinary black powder, but also the smokeless, gray, or white powder. This latter is composed of nitroglycerin, and perhaps other high explosives, modified or reduced by different combinations. The phenomena attendant on its use are exceedingly interesting, and differ greatly from those of the ordinary black powder, especially in the matter of marks, burnings, and powder stains. The prominent feature of the smokeless powder is its rapidity of action. It explodes almost instantaneously, or, at all events, three times as rapidly as the common black powder. Its greatest effect is exerted on the solids in direct contact with it, and often downward if on the ground. Hence its power as a blasting agent. I once witnessed the effect of the explosion of a large pile of dynamite which was awaiting transportation. A deep hole was excavated in the ground under it, while a flimsy scaffold, erected for chemical tests, only a few feet away, remained undisturbed, with unbroken glass retorts and testing appliances. A house a quarter of a mile distant was greatly damaged. Death was all around.

When gray powder, the "smokeless" powder of commerce, is fired from a cartridge, it will produce a circular powder mark, but not the brand, as shown in the illustration,—or at all events, only to a very slight degree, scarcely to be appreciated. Indeed, if there is any, it is in a reversed position,—that is, away from the hammer, for it is in this direction that the effects of the explosion of the smokeless powder are manifested. Neither is there much evidence of the tattooing from unburned or partially burned powder grains. The explosion of smokeless powder, its conversion into gases, is fully three times as rapid as that of the black powder. In the former there occurs practically instantaneous and complete combustion; in the latter, a relatively slow combustion. In a dark-

ened room it is possible to recognize a face from the comparatively prolonged flash of the black powder, as you may readily notice by experimentation. Not so when the smokeless powder is used, when the explosion is so complete and the combustion or flash so quick. In the targets from smokeless powder, you will notice the peculiar circumferential black discoloration and its smooth surface, but the scorched and brownish tongue-shaped charring characteristic of the explosion at short range of the ordinary powder is conspicuously absent.

From these brief remarks it will, I think, be quite evident that, in venturing any professional opinion in a medicolegal gunshot case, the peculiar action of the gray, white, or smokeless powder should be considered. Its effects, in striking contrast with those of the ordinary black powder, as regards rapidity and completeness of explosion, brand, flash, and powder marks, should always be borne in mind. Otherwise an erroneous opinion may be formed as to the character and position of a weapon from which a given shot was fired.

THE TREATMENT OF FRACTURES AND DISLOCATIONS IN RELATION TO SUITS FOR MALPRACTICE.[1]

BY JOHN A. LEWIS, M.D.,
Of Georgetown, Kentucky.

Gentlemen,—The subject of this paper should interest each of you equally, whether you be an humble practitioner of medicine, unostentatiously performing your arduous labors in some remote and unknown village or in more quiet country, or whether you be some surgeon of world-wide fame in a crowded metropolis who numbers among his patients the rich and the *élite*. It touches each alike in two most vital points, the reputation and the pocket.

A suit for malpractice may follow the treatment of any ill to which human flesh is heir, in any of the various departments of the practice of medicine; yet the majority of these damage suits fall to the lot of the surgeon, and most of them result from either real or imaginary deformities which occur in connection with the treatment of fractures and dislocations. For this reason I have selected the limited field of the treatment of these injuries as typical of the entire subject of suits for malpractice, rather than the discussion of the question from the standpoint of the general practitioner, which would manifestly be too extensive for this occasion.

This is an age of damage suits; indeed they seem to be the prevailing fad. There was a time in the dim past, the good old days of our fathers and of the stage-coach, when a suit for damages for any cause was a thing almost unheard of, and the man who brought one was laid under suspicion as being actuated by motives other than the highest. In those days men took their lives in their hands, assumed their own personal risk, and went forth like knights to take their chances in the rough battle of life. But not so now; the modern gladiator who steps boldly into the arena of life, espe-

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[1] Read before the Kentucky State Medical Society at Paducah, Ky., May 7, 1902.

cially if the battle be extra-hazardous, is nerved to the combat with the reflection that he will hold some one for pecuniary damages for any black eyes or broken bones which may fall to his lot in the encounter. The passenger on the railway train who receives the slightest injury in being transported from one point to another is sure to institute suit against the railway company as soon as he recovers his breath sufficiently to call a lawyer through a telephone.

The employés on the railway, in the mine, in the foundry, in the factory,—indeed those employed in any capacity whatever,—have ever present with them the hope of reimbursement for any injury they may sustain in any of their various pursuits. Even the boy who drives the village doctor in his morning rounds (necessarily exposed to contagion) has high hopes for damages in case he breaks out with the measles, complicated with an attack of otitis media, while following his calling with the full consent of his family.

But, above all, the doctor, the custodian of life and limb, comes in for a double share of trouble along these lines. He is held to the strictest accountability for sins of omission and commission. If his most delicate and difficult task is not performed to the entire satisfaction not only of the patient and his family, but also of the entire neighborhood, he may very promptly be subjected to a suit for malpractice. It seems strange, and to say the least of it hard, that the men who above all others live lives of self-sacrifice, ministering to the sick and the suffering of their respective communities, who freely give their services to the poor, day and night, without money and without price, should be ever held under suspicion and the strictest surveillance, while for the least failure, real or imaginary, they are liable to be set upon by the harpies of the law.

It is a well-known fact that the charity patient or the man who (though not an object of charity) never pays nor expects to pay a bill for medical services is the man who more frequently than any other seeks redress at the hands of the law for unsuccessful treatment by the surgeon. This is one of the many ways which the "dead-beat" adopts to escape the payment of a medical bill; by bringing suit he hopes to effect either a compromise or to offset your bill by a counter one. It would be well for every physician to remember that the law holds him to the same strict responsibility in the treatment of the charity patient as it does in the treatment

of the well-to-do. We must guard ourselves well when dealing with this class of patients.

There are some people in this world so in love with money that they would be willing to sacrifice an arm, a leg, an eye, or anything about the corporeal outfit which they could conveniently spare, for a reasonable financial gain. So, whenever they get an injury, and the surgeon fails in any way to secure a better member than the original one, they seize upon the flaw, real or imaginary, as a cause for legal action. But there is another cause for these suits of which I must speak, and I do it with the blush of shame on my cheek; it is jealousy, jealousy between brother physicians, physicians who above all others should be true to one another. By some of the mutations of time, a case which one physician has treated for a fracture, a dislocation, or other injury comes under the observation of a brother physician; by innuendo, by unfavorable comment upon the result, or by some faint praise, he damns his brother. The suggestion having been made, the "injured" party loses no time in consulting a lawyer, and they get up a case with the distinct understanding between lawyer and client that they are to share alike. A lawyer has been found to prosecute the case against the surgeon, and (who would believe it?) occasionally a surgeon is found who is willing to be a pliant witness in the hands of the lawyer. Truly, the "ox knoweth his owner, and the ass his master's crib." *O tempora, O mores!* "Let my tongue cleave to the roof of my mouth," and "my right hand forget her cunning," in the hour when I shall appear as anybody's witness save the truth's!

Do not understand me to say that there are no just suits for malpractice against physicians or surgeons; far from it. Criminal ignorance, neglect, unfaithfulness, in consequence of which the usefulness or happiness and comfort of a human being is destroyed, should not go unpunished. Neither would I have you believe that the majority of our patients are not only ready but anxious to find some flaw in the treatment of their cases which would justify them in bringing suit for damages. On the contrary, I gladly and cheerfully bear testimony to the generosity, nobility, and appreciativeness of the majority of those who call upon our profession for treatment for their many ills. But there are those, and they are not a few, who are glad of an opportunity to hold the physician responsible for some real or imaginary defect in treatment. They seem

entirely oblivious to the difficulties confronting the surgeon, and do not recognize the fact that failure is not impossible in any case, and that no surgeon can positively guarantee perfect results even in uncomplicated cases. The public sadly needs education along this line. Patients ought to be made to understand how serious, difficult, and delicate a matter it is to treat even a simple fracture or dislocation, and how much more difficult to treat a serious and complicated one; how hard to make out the exact anatomical relation and position in a bruised, swollen, and distorted limb, whether the injury is a dislocation or a fracture, or both, and how difficult to return accurately to its normal position and to maintain in position after the injury has been treated. They ought to understand that a surgeon is mortal, and like other men is fallible. The wonder is, when we contemplate the difficult surroundings, that failure does not occur oftener than it does.

There are cases of simple fracture (notably oblique ones of some of the long bones) in which failure may follow treatment although the surgeon may have done everything which skill and faithful attention dictate. He may have recognized perfectly the form of injury, may have reduced and perfectly adjusted the fragments and skilfully supported the limb by properly applied splints, may have carefully and faithfully watched the injury through the entire course, and yet upon that supreme day, that day of suspense to the surgeon when dressings are removed, he may find the result is not what it ought to be and not what he expected. And yet the surgeon should not be held responsible for the failure, unless a man should be held responsible for an impossibility. I know this is putting the matter strongly, but I will state my reasons.

Remember, if you please, how hard it is to maintain an oblique fracture in position after it has been accurately adjusted and supported after the most approved manner. We frequently have violent and sudden muscular contraction, nearly jerking the patient out of the bed; this often occurs while the patient is asleep, and attendants are required to sit by him constantly to control this as far as possible. It is a well-established fact that fractures of bones have occurred from muscular contraction alone. The patient cannot lie in one position throughout his entire treatment, but must be turned from time to time; but any movement may excite mus-

cular contraction, and may displace these oblique fractures after they have been perfectly reduced and splinted.

We are dealing with flesh and blood, not with a buggy shaft or a wooden leg; if with either of these, any tyro could so bind them up as to warrant results; but we are dealing with a human limb, composed of flesh and blood, and nerve and tendon. The supports and bandages must be just firm enough to hold, and yet not interfere by pressure with circulation or nerve. The limb may swell or it may shrink under pressure. No human tact can meet all these delicate indications in every instance; it is beyond human skill and ingenuity, and no man is infallible. He may do all in his power, all that skill dictates, and yet be disappointed. It may have been rightly adjusted, rightly and skilfully treated, and still have gone wrong underneath the dressing.

I will mention one other matter which the patient and his family should remember in making up their estimate of the surgeon's responsibility in treatment of these cases. The surgeon is not in the case of his own bidding. He has been selected by the patient or his family for his already known ability, or after inquiry and investigation. Those employing him know his caliber, whether or not he has any special skill or adaptation in treatment of these injuries. They had at their beck and call every surgeon from New York to San Francisco; it was simply a matter of money and convenience,—they could have paid their money and taken their choice. But they may be willing to assume the risk, and trust the case to the mediocre surgeon, rather than undergo the outlay of money; if failure follows, who is to blame? Certainly not the surgeon, unless gross neglect and carelessness can be proved. The truth is that the patient assumes the risk, and if failure follows he should blame no one but himself.

In consideration of the many difficulties which confront the surgeon in the difficult and responsible task of treating fractures and dislocations, and the possibility of a suit for malpractice in consequence of unsuccessful results, what shall we do? And how shall we proceed to protect ourselves?

In the first place, no man who has not some special skill and training in this direction should ever undertake the treatment and care of this class of cases. If, however, you undertake to treat these injuries, then make up your mind to give those who fall into

your hands your most skilful and painstaking and constant attention, from start to finish.

In my judgment, the time to make a complete and thorough examination of your patient is upon your first visit, at the time of the accident. In the majority of cases the conditions are better for making an accurate diagnosis and your opportunity more favorable for reposition and adjustment of the injury then than ever afterwards. On your first visit you will find your patient willing and anxious to take an anæsthetic while undergoing an examination; in a few days it may not be so. The muscles are more relaxed, there is less effusion, less induration, less fixedness, at the time of injury than ever again.

After you have examined the patient and made your diagnosis it is all-important that you should be perfectly frank with the family; tell them the exact nature of the injury and just what they may expect, hold back nothing, do not be over-confident of results, and guard your prognosis well. If possible have some honest practitioner or, better still, some intelligent and respectable layman present, to hear your statement when made, and be sure you state the facts so plainly and so strongly as to impress the situation upon their minds. If you are uncertain about the form of the injury or about the proper method of treatment, call at once for a consulting physician, and tell the family you are unwilling to assume the entire responsibility. In selecting your methods of treatment, use only those that are approved; make no experiments. The courts have held that a physician is liable for damages in case injury comes to the patient from the use of some new and untried method, although the object and effort were to do good.

It may be useful and helpful to review very briefly the methods of examination and treatment of some of the more common fractures and dislocations the treatment of which is most frequently followed by unsatisfactory results.

Perhaps the most common fracture to which the human body is liable is that of the forearm, generally the radius. The fact that the hand and arm are ever on guard, standing between us and a thousand dangers, will account for this frequency. The fracture of the lower extremity of the radius is the one most frequently followed by imperfect results, and consequently most liable to bring trouble. The injury is known as Colles's fracture, and is easily

recognized by its resemblance to a silver fork. It may occur at any age, but is most frequent in advanced life. The treatment of this fracture, especially in the aged, is often followed by deformity, which generally consists in an anterior prominence of the lower end of the radius, coupled with anterior and lateral displacement of the ulna, stiffened fingers and wrist, and limited use of the hand.

In my opinion, the frequency of deformity after this particular fracture is due to the fact that we rarely get a perfect reduction of the fractured end of the radius; the fracture may be an impacted one, the fragments may be small, and so wedged in between the carpal bones and the lower end of the radius as to render replacement difficult. Besides, the lateral ligaments and structures which bind the ulna to the radius are so torn and divulsed that they never again hold the ulna and radius in close apposition. Another reason why a more accurate adjustment of this fracture is not secured may be that an anæsthetic is rarely given in adjusting the injury; it is hardly thought necessary, the seeming replacing requiring only a moment, besides which many of the patients are old and we shrink from administering an anæsthetic. The proper reduction often requires considerable direct force and pressure, and we fall short of this when the patient cries out with pain. To secure satisfactory results, perfect reposition of the fragments before applying splints is imperative.

The deformity and swelling of the fingers and hand occur in the aged, and are the results of protracted confinement in splints, coupled with the enfeeblement of the circulation. This must be avoided by allowing splints to extend only to the fingers, leaving the latter unconfined, and by keeping up motion in them as far as possible from the day of injury until the splints are removed.

Next to the wrist perhaps injuries at the elbow are most liable to give trouble; mistake in diagnosis and treatment may result in imperfect and unsatisfactory results. This joint comprises several bones, processes, and ligaments, making it very complex. There may be a dislocation of the joint, a fracture of the condyles, fracture of the lower end of the humerus, fracture of the olecranon, or fracture or dislocation of the head of the radius. Great care must be exercised to avoid error in diagnosis, which would lead to grave results. In your examination under an anæsthetic locate the bony prominences, the condyles, the olecranon, its fossa, and the head of

the radius. By careful manipulation see that the joint flexes and extends, and that the head of the radius is in place. If you have a fracture, crepitus will indicate it; if dislocation, reduction lessens the deformity, the joint will play. Dress all injuries at the joint, except fracture of the olecranon, with elbow flexed. Whether fracture or dislocation, you may certainly predict immobility and limited flexion of joint for some months, but wonderful resumption of natural function follows prolonged use in these elbow injuries.

In injuries of the shoulder we must again be very careful to make a clear diagnosis; fracture and dislocation must be differentiated. Locate carefully the bony landmarks, as these must be your guides. The acromion, the coracoid processes, and the head of the humerus, all in proper relation, make up this joint, and any deviation must be taken into account. Fractures of the long bones are all easy of diagnosis, but the surfaces are not always easily kept in apposition after reduction, especially in oblique fractures. When a fracture of any of the long bones is first inspected after splints are removed, frequently there will be considerable seeming deformity at the point of fracture, especially in fracture of the shaft of the femur, but much of this deformity is more apparent than real. The large amount of provisional callus always thrown out around the point of injury, as well as the fact that the limb is very much reduced in flesh from pressure and disuse (only skin and bone are left), will account for this; time, and flesh on the shaft of the bone, will help the appearance wonderfully. Several times in my life I have been badly frightened at the first look after dressings have been removed.

Injuries at the hip-joint are always serious and sometimes obscure, and mistakes are readily made in this region. The difficulty lies in the differential diagnosis between the varieties of dislocations and in distinguishing between fracture and some of the forms of dislocation. Let it be remembered that a fall on the hip in an elderly person, accompanied by pain, helplessness, eversion of the foot, shortening, and prominence of the trochanter, may be set down as a fracture of the hip, whether we have crepitus or not. It should be treated as a fracture; the confinement necessary to demonstrate whether we have a fracture or not is nothing as compared with the trouble which might arise if a mistake were made. Long confinement and imperfect recovery may be certainly predicted, and lame-

ness for life is nearly always the result; death frequently follows these cases in the very old and feeble. In the young, dislocations must be looked for after falls from horses and wagons or from any considerable height; the diagnosis is generally easy, but such cases have been overlooked and followed by lifetime lameness. In these hip injuries never fail to call in help if you are in doubt, as an oversight resulting in the permanent disability of young persons will never be forgiven.

This paper would be incomplete if I did not mention the use of the X-rays in the diagnosis of these obscure injuries. This means of examination is certainly the most important advance of the age along this line, and is destined to come into general use; so far it has been largely in the hands of the specialist. The expense of the outfit and the skill necessary to its application have limited its use. But we should never fail to avail ourselves of its aid in diagnosis in all obscure injuries whenever practicable. Suits for malpractice have followed its use, parties claiming to have been injured by too long or too powerful exposure to the rays. We hope this source of trouble will disappear with a more perfect method of application.

Since any one of us is liable at any time to be confronted by a suit for malpractice, the question as to what shall be done to avoid trouble becomes a vital one. Many physicians and surgeons advise putting all property out of your hands, but no man who does this can maintain his financial standing in any community. Others advise taking out an indemnity policy in some insurance company, in which case for an annual premium the company agrees to defend you to a certain amount in any damage suit which may arise during the existence of the policy. Just how efficiently and how faithfully they might defend the suit I am unable to say. Whether you resort to any of these expedients or not, I am sure you will find the following summary of this paper helpful. (1) Do not undertake to practise surgery unless you have some training and skill in that direction. (2) If you undertake it, leave nothing undone to secure success, from the day of your first visit to your last. (3) Never warrant results; let it be clearly understood that failure is not impossible even in uncomplicated cases after the most faithful and skilful treatment. (4) Make your first examination just as carefully and thoroughly as possible under the circumstances; make a

plain and truthful statement as to result of your examination, of trying nature of injury, etc., to the family, guarding your prognosis well. Make this statement, if possible, in the presence of some honest physician or, better still, some intelligent layman. (5) Let the patient and his family feel that in calling you it is presupposed that they know your ability in work in this line; if they are unwilling to assume the risk of the final results, they ought to call some one else. (6) After your first examination, if you are in doubt as to the diagnosis or as to the plan of treatment, call a consulting physician at once. (7) In the treatment of charity patients and dead-beats, be especially on guard to protect yourself; remember the law holds you to the same accountability in treatment of a charity patient as in treatment of the rich. (8) Use only approved methods of treatment; leave the experimental treatment to the hospitals. (9) Treat your brother physician exactly as you would have him treat you; never by word, or act, or look lend countenance to the suggestion of a damage suit, it matters not whether the physician be your personal friend or not. Express no opinion as to an unsuccessful result in any case treated by another physician unless he be present. (10) If, in spite of your best efforts to avoid a suit, one comes, never compromise or pay hush-money, but fight to the death. Every physician should always stand ready to go to the assistance of his brother physician, with his time and money if necessary, in defending one of these suits for malpractice.

INTERNAL PILES; THE CLAMP AND CAUTERY OPERATION; TUBERCULOUS FISTULÆ.

CLINICAL LECTURE DELIVERED AT THE NEW YORK POLYCLINIC HOSPITAL AND SCHOOL FOR GRADUATES IN MEDICINE.

BY JAMES P. TUTTLE, M.D.,

Professor of Rectal Surgery at the New York Polyclinic, etc.

Gentlemen,—Our first patient complains that his rectum "comes down," as he says, and of frequent hemorrhages at stool. From the fact that he has suffered considerable inconvenience in defecation and is of a constipated habit, it seems probable that he is suffering from piles. On examination of the anal region, we find upon the external skin signs that point to antecedent inflammation of the rectum. Here and there, too, are the hypertrophied papillæ, each presenting a little split, characteristic of previous ulceration. In exploring the rectum, I find that there is a tendency of the mucous membrane to prolapse. The internal rectal tissues are rather dry, and the bowel lacks something of the velvety feel of the normal mucous membrane. As my finger passes higher up, I find what I believe to be an adhesion of the appendix to the rectum. Under my finger there is a rounded body that I can roll against the promontory of the sacrum. This imparts to the touch the sensation of a thick, cordlike structure without notable induration. I have on two occasions been able to detect a similar adhesion of the appendix to the rectum. The inflammation that caused the adhesion limits rectal peristalsis, and this functional derangement usually gives rise to constipation, which is not infrequently complicated by piles.

We shall not advise operation for the appendiceal adhesion unless special symptoms arise that point to the appendix as their primary cause. As I stretch the external sphincter a rosette of internal piles appears. The chief anterior hemorrhoid in this case is on the patient's left and not on the right, as is usual. The sphincter in these cases should be dilated very slowly lest the mucous membrane be ruptured. At least three minutes should be allowed for

this procedure. More rapid dilatation will not only occasion the patient considerable discomfort after the operation, but, by causing fissures in the mucous membrane, opens the door to infection. Although I have stretched the sphincter thoroughly, you see how promptly it contracts and closes the anal orifice as soon as I remove my finger. Stretching the sphincter does not cure fissure of the anus by putting the sphincter muscle at rest. Unless an accidental rupture of the muscular fibres occurs, this muscle completely regains its tone in twenty-four hours. The sphincter is not long enough at rest for the fissure to heal; the favorable action of dilatation must, therefore, be explained in another way. Apprehension lest the sphincter may not promptly recover its tone after having been thoroughly stretched is groundless.

You know my preference for the limited excision and suture operation for the removal of piles. To my mind this is the ideal method in certain forms of this disease. It leaves no open wound, and is attended by no danger of post-operative suppuration nor of stricture from the contraction of cicatricial tissue. Although the case before you is suited to the excision and suture method, I prefer to demonstrate to you the use of the clamp and cautery instead, because this operation is the simplest and easiest for the general practitioner. You notice that I stretched the sphincter before cleansing the operative area. This is a practical point of some value. For if the washing were done before the sphincters were stretched the site of operation would be soiled by mucous discharges and fecal matter, and a second cleansing would be necessary.

The first step in the operation is to grasp the pile with forceps and cut through the skin with scissors at the mucocutaneous junction. By inserting the clamp in the groove thus made, which is not deep enough to cause much bleeding, burning the sensitive skin is avoided. The forceps with which each pile is grasped has an oblong bite, and over it the clamp is applied in such a way as to grasp the pile parallel with the long axis of the intestinal tube. This causes the cicatrix resulting from cauterization to be parallel with, and not to encircle, the gut. Danger of stricture from cicatricial contraction is thus obviated. The technic of this method is simple. The clamp, grasped by the right hand, should be brought down over the forceps. In this way not only is the pile caught

lengthwise, but the upper part of the gut is seized by the part of the forceps where its jaws are most closely approximated, and the greatest crushing force is exerted at the upper part of the mucous membrane where the vessels are largest. This minimizes the only risk of the operation—hemorrhage. When the forceps is applied, the screw should always be run down so as to hold the jaws of the instrument firmly in position. This is not an unnecessary precaution, although it involves considerable loss of time. The only serious bleeding I ever met after the use of the clamp and cautery occurred in a case in which I failed to use the screw. My assistant in handing me the cautery touched me on the arm with the hot part of it. I naturally relaxed my grasp upon the clamp, and the tissues slipped back into the rectum. As the parts had been sufficiently crushed to prevent bleeding for the moment, I could discover no bleeding points, but I could not satisfy myself that all the vessels were closed. I dressed the wound and put the patient back to bed, calling the attention of the house surgeon to the possible occurrence of secondary hemorrhage. About three hours afterwards the patient was compelled to have a movement of the bowels, at which he passed about twenty-four ounces of clotted blood. The hemorrhage was checked by firmly packing the rectum with gauze. This was a lesson to me, and I pass its warning on to you.

Each of the piles having been caught in the way that I have described, the projecting portion is cut off, leaving about one-fourth inch of tissue above the clamp, and this stump is seared with the Paquelin cautery. Cauterization seals all the lymphatics and prevents absorption of infectious material passing through the rectum. The burnt tissue breaks down and is discharged some days after the operation, but no infection occurs. After the removal of the piles, a dressing of orthoform is applied. This is not only antiseptic, but it serves the very useful purpose of lessening pain. The rectum should be packed rather firmly with gauze, which should be allowed to remain for about twenty-four hours. No subsequent packing of the rectum is usually needed, an external dressing only being required for the protection of the bed-linen and of the clothing.

After the first dressing orthoform should not be employed. If used too long this substance has a tendency to produce dermatitis. In parts granulating after cauterization it also causes a prolon-

gation of the ordinary period of inflammatory secretion, and the tissues are prone to slough more than under less powerful remedial agents. At subsequent dressings, then, the outer parts should be washed with a weak solution of bichloride of mercury, not stronger than 1-5000, or with a boric acid solution.

Patients who have been operated on for piles do not need to be catheterized as frequently as is generally believed. It is true, however, that disturbance of the peripheral nerve endings around the anus does, in some instances, produce a reflex spasm of the muscles that control urination. In these cases, which are, however, comparatively rare, the catheter will have to be employed. The reason why most men are unable to void urine freely is that they are asked to urinate lying down. For some this is extremely difficult. Many, too, cannot pass water into a urinal, especially under the psychical excitement of a hospital ward. There is no reason, however, why a patient operated on for piles should be required to maintain the recumbent position. After six hours or so he may be permitted to stand up in order to urinate, and then usually there will be no trouble in the performance of the act. When this rational precaution is taken it will be found that not more than one patient in ten will require the catheter.

This point should be insisted upon, as serious injury to the bladder may be done by the catheter even in the most careful hands. A non-specific urethritis, as the result of injury to the mucous membrane of the urethra, is not a rare accident if frequent catheterization is practised. Even though the catheter be absolutely sterile when used, infectious material from the urethra which has an abundant flora will cling to it and may be carried back into the bladder and cause cystitis. Comparatively few men are without what the French in picturesque phrase call a " urethral past." Even though there is no discharge from the urethra, there may be an inflammatory focus somewhere along its course.

Cystic disturbance may persist for months after the removal of hemorrhoids, though there had been no bladder trouble before the operation. I do not think the stress I lay upon this point is unwarranted, for I have had unfortunate experiences, and I wish to warn you. When the catheter is employed, voluntary urination should be encouraged in every way. Above all do not establish the catheter habit by suggestion. The patient should not be told that

catheterization may have to be done for several days after the operation. When the catheter is used for the first time, tell the patient that it will probably not have to be employed again, the idea being to produce a state of mind favorable to voluntary urination.

The clamp and cautery operation requires practically no after-treatment. The packing is removed in from twenty-four to thirty-six hours, and the patient is given a mild laxative like cascara. Drastic saline purgatives should not be used; they add to the patient's discomfort and increase the liability to spasm of the rectum. If the spasm of the rectal tissues be excessive, and the patient dreads the pain caused by defecation, a moderately large rectal bougie should be passed in order to straighten out the rectal tissue. After this has been once effectually done, it is not usually necessary to repeat it. The average length of time that the patient must keep his bed is about four days. He can always be assured that he will be back at work within a week or ten days. At the end of twenty days, as a rule, all pathological discharge from the rectum will have ceased. Patients who are not sensitive to discomfort are often able to resume their occupations in four or five days. I operated upon a doctor last year under nitrous oxide gas. The operation required just three minutes. Six minutes after he had taken the gas he was back in bed talking to me. He had been bothered for many months, and was rejoicing over the fact that he was at the end of his trouble. He suffered from a good deal of spasm during the afternoon, due, probably, to the fact that the dilatation of the sphincter had been rapid. His discomfort, however, was not sufficient to keep him from driving home, some thirty blocks, the same day. The next day, after having had a rather easy movement, he came down to his office and kept his regular office hours. He continued his professional duties without difficulty thereafter. Not very long ago I had a still more amusing experience with a doctor from the South who had been suffering from piles for some years, upon whom I did the clamp and cautery operation. He was a large, strong, healthy man, and I anticipated no difficulty at all after the operation. I called to see him about four or five hours after the operation, and was surprised to find him absent from his room. The next day he came to my office. I asked him where he had been the afternoon before, and he replied that, as he was feeling very well and had no discomfort, he had taken a walk.

Further inquiry elicited the fact that he had walked to the Battery and back, a distance of seven or eight miles.

As I have said, the cautery operation is practically never followed by infection. Cauterization seals the lymphatic channels and destroys the bacteria that may be present in the tissues operated upon. This operation is the ideal one for the country surgeon who has to operate in private houses at a distance from his office. Though the operation may have been performed ten miles from home, the doctor can leave his patient with perfect confidence that nothing will happen to mar the aftercourse of the case. If he be unable to see the patient for a day or two after the operation he may simply direct the removal of the packing from the rectum in thirty-six hours and the administration of a mild laxative. If the patient live in an out-of-the-way place, the doctor need not necessarily visit him again, but may wait until the patient is able to come to his office. Stricture does not occur after this operation. The only serious sequel that in rare cases follows is protracted ulceration. This usually depends not on lack of asepsis during the operation, but upon the unfavorable condition of the patient's health. If the general vitality be low, the ordinary bacteria in the intestine may multiply in the sloughing tissues and cause discomfort for a time. As a rule, however, this complication need not be anticipated in an ordinarily healthy patient.

Only four instruments are required for the operation,—scissors, hemorrhoidal forceps, the clamp, and the Paquelin cautery. Of these only the Paquelin cautery is somewhat expensive. Practitioners should have it, however, if for no other purpose than to use it is a means of counter-irritation in muscular rheumatism. I have had considerable bother from muscular rheumatism myself, and I have obtained more relief from ironing with the cautery than from any other method of treatment. For those who have suffered from this annoying affection and have obtained relief in this way, no other therapeutic method is so satisfactory.

TUBERCULOUS FISTULÆ.

There are two types of tuberculous affection that occur in the neighborhood of the rectum. These are localized and diffuse tuberculosis. Both are liable to be complicated by external or internal fistulous openings and communications between the gut and the

skin. I have a case to show you to-day that illustrates very well the type of diffuse tuberculous infiltration of the circumrectal tissues. Our patient, a man thirty-five years of age, has tuberculous deposits at the apices of both lungs, and his general physical condition is unsatisfactory. When he came to the hospital a few days ago, he presented the characteristic symptoms of a perirectal abscess.

The abscess was opened and drained. I did not explore the abscess cavity very carefully because the patient's condition was such that we did not care to keep him under narcosis any longer than was absolutely necessary. Notwithstanding careful drainage, the abscess refused to heal and continued to discharge more pus than its apparent size would account for. Subsequent examination showed not only that there was an opening from the abscess into the bowel, but that there was also a second communicating with a larger abscess cavity located above the first one. It was from this second abscess cavity that the excessive discharge of pus came.

It has been the custom to recommend the free opening of abscesses like these without any special direction as to the manner in which this should be done. No greater mistake could well be made. The use of the knife in such cases exposes the tissues to further infection, and not infrequently febrile movements occur shortly after the incision of an abscess wall, when the patient's temperature was previously normal. Needless to say in the debilitated condition of patients with lesions fever proves rapidly exhausting.

As the result of the widespread discussion of the dissemination of tuberculosis that followed the meeting of the British Congress on tuberculosis last August, one conclusion forces itself upon all minds. This is that nature, by throwing a wall of fibrous tissue around tuberculous foci, is able to isolate collections of tuberculous material and prevent the extension of the disease beyond certain limited areas. The walls of a tuberculous abscess cavity are composed of three layers. The first or innermost layer consists of granulation tissue. Microscopic examination of this part of the wall or of scrapings from it show that it is full of tubercle bacilli. The second layer is composed of embryonic connective tissue in which a few tubercle bacilli are to be found. The bacilli are, however, much less numerous than in the inner or granulation layer. It is in this part of the abscess wall that giant cells are most abun-

dant; many of these large multinuclear cells contain tubercle bacilli, some of which are in a more or less disintegrated condition. Here phagocytosis—nature's great protective mechanism against infections—is most active. The bacilli that succeed in finding their way beyond the granulation tissue layer of the abscess are englobed and digested by the phagocyte cells. It is probable that most of the tubercle bacilli in this layer have been robbed of their virulence by the action of the digestive ferments whose presence has been demonstrated in the leucocyte cells of this part of the abscess walls. The third layer of the abscess cavity consists of fully formed fibrous tissue. Fibrinous tissue contains very few blood-vessels and practically no lymphatics, and forms an effective barrier to the passage of bacilli of every form. After the abscess has been evacuated this fibrous tissue contracts and obliterates the abscess cavity, encapsulating whatever cheesy material remains, and gradually abstracting the fluid constituents of the diseased tissue, thereby rendering the bacilli innocuous. This protective mechanism of nature may be disturbed if tuberculous abscesses near the rectum, especially those which communicate with rectal fistulæ, are improperly treated. In order to imitate nature's methods, such abscesses should not be opened with a knife, which leaves gaping lymphatics to absorb infectious material, but with a cautery, which destroys infectious material in the neighborhood of the wound, and sears the blood-vessels and lymphatics severed by the incision. The Paquelin cautery should not be allowed to get beyond a dull red heat.

In the case of our patient, the wall of the secondary abscess cavity which is situated above the one which was evacuated by our operation of the other day, is slowly burned through in such a way as to secure thorough drainage. It is not unusual to find a secondary abscess cavity of this kind connected with a cavity below it by a narrow neck through which satisfactory drainage is impossible even though the opening in the lower abscess cavity be kept patulous. Tuberculous abscesses in the neighborhood of the rectum must be treated with every antiseptic precaution. The slightest negligence may result in secondary infection, which is often much more serious than the original tuberculous affection. As a matter of fact most specialists in tuberculosis now claim that it is the secondary infection in tuberculous processes, rather than the primary disease, that entails the gravest consequences for the patient.

After having made an opening with the cautery large enough to secure thorough drainage, gauze dusted with orthoform is introduced into the abscess cavity. The dressing will have to be renewed every other day, and orthoform may be dusted over the tissues in order to relieve pain, for which purpose it answers admirably. The important part of the after-treatment, however, is the improvement of the patient's general condition. To this end we encourage his going out into the open air as much as possible and endeavor to maintain good digestion.

To-day our patient will remain in bed. But to-morrow I shall suggest his going down to the docks or into the park during the sunny hours. He must have an abundance of pure, fresh air and must not be allowed to remain indoors as patients of this class are prone to do. Indoor life usually means chronic suppuration for these cases. Diffuse tuberculous processes around the rectum absolutely refuse to heal properly unless the general system can be built up sufficiently to increase vital reaction.

We must always distinguish tuberculous fistulæ from fistulæ in the tuberculous. Tuberculous fistulæ are always localized lesions, consisting mainly of narrow fistulous tracts. They are walled off from the surrounding tissues by the three layers above described, they are not very dangerous, and cause but little inconvenience. While many suffering from chronic tuberculosis have fistulæ, these are of the limited variety, and rarely demand surgical interference. The tendency in advanced life to the formation of fibrous tissues retards the spread of tuberculous foci. As a consequence of this, while tuberculosis is not infrequent, it is rarely fatal in old people. Of the bodies of the aged that come to autopsy in almshouses and charity hospitals about eighty-five per cent. have tuberculous foci, but in only fifteen per cent. is tuberculosis the cause of death. In some individuals, in earlier years, there is a distinctly greater tendency to the formation of connective tissue than in others, and in the former tuberculosis runs a slow course.

When nature has surrounded tuberculous fistulous tracts with an adequate wall of fibrous tissue, it is not well to interfere with the protective mechanism thus provided by active treatment. Patients sometimes have a slight watery or opalescent discharge from a small, more or less tortuous fistula not far from the anus, that causes but little real inconvenience. The knowledge of the exist-

ence of the fistula, and the fact that it tends to soil their clothing occasionally are the principal grounds of complaint. In my judgment, such cases should be left absolutely alone. The only operation likely to be of lasting service in such cases would be the excision of the fistulous tract and the inflammatory products adjoining it throughout its whole course, in order to obtain primary healing of the healthy tissues beyond. This operation, although difficult and tedious, is, however, perfectly feasible, and in simple cases may be performed without much difficulty. Ordinarily, however, the symptoms from which the patient suffers are not sufficiently annoying to justify the extensive interference that would be required, especially as the surgeon may not be able to remove all infectious material, or to guard against possible infection of the tissues on which he operates with resultant recurrence of the fistula or of some inflammatory process. Surgical interference with sluggish tuberculous foci that exist in these small fistulæ may easily light up extensive ulceration. If the knife be used the lymphatics are laid open, absorption of infectious material follows and some extension of the inflammatory condition is inevitable. If left alone such fistulæ may exist for many years without the occurrence of serious complications. I have histories of the persistence of such lesions for twenty, even thirty years without dangerous or even annoying consequences. *Per contra,* an acute ulcerative process can cause serious trouble for a long time and may even shorten life.

In diffuse tuberculous processes in the neighborhood of the rectum, we have quite another condition to deal with. Usually such cases are not due to simple tuberculous infection. Secondary infection has taken place, and it is to the combined infection that most of the serious symptoms of the condition are due. In such cases thorough drainage must be provided, collections of pus must be evacuated, and abscess cavities made to heal from the bottom.

In conclusion, then, I may say, that when you meet cases of more or less straight weeping fistulæ in old patients, tell them gently but firmly to bear the ills they have, rather than fly to those they know not of. Endeavor to secure reasonably regular evacuation and soft stools, but leave the fistulæ alone as far as operation is concerned. Radical surgical cure is, of course, the ideal treatment in these cases, but, like most ideals, it is rather difficult of attainment, and it is better, as a rule, to be safe than to be sorry.

OBSTIPATION; HEMORRHOIDS; FISSURE ANI.

CLINICAL LECTURE DELIVERED AT THE CHICAGO POLICLINIC.

BY J. RAWSON PENNINGTON, M.D.,

Professor of Rectal Diseases in the Chicago Policlinic, Chicago, Illinois.

GENTLEMEN,—The first patient I show you this morning is one you saw at our last clinic, whose history pointed to obstinate constipation, or, perhaps more correctly speaking, obstipation. You will also remember that he gave the history of a condition which we call mucous colitis. It is my intention to make a high proctocolonic examination of this patient, and to apply one or more of my automatic valve clips to the *plica transversalis recti,* should I deem it necessary. This is best and most safely accomplished by first firmly and securely retaining the patient in the knee-chest posture or its equivalent. While the patient is being anæsthetized, I will briefly explain to you the mechanism of a small portable table, which I designed especially for this class of work; at the same time, it is suitable for all classes of surgical work and urethral catheterization. It can be tilted directly backward to an angle of more than forty-five degrees. There is no other table, to my knowledge, that can be tilted to such an extent, and yet this extreme position is frequently desired. You notice [shows table] that it is quickly and easily adjusted to any position, and securely locked therein. The upright bar on the side is to support the patient's knees in making sigmoid and high rectal examinations. The strap is to be placed over the patient's shoulder to prevent his slipping. The bracket, as you see, is attached to the side of the table, and is readily thrown into any position desired. The lamp for illuminating the ballooned bowel is the ordinary bicycle acetylene-gas lamp. The table is readily folded into a small package convenient for removal.

The patient being anæsthetized and properly adjusted on the table, we will proceed with the examination. You will notice that I remove the obturator as soon as the speculum enters the pouch of the rectum. I can then continue the further introduction of the

speculum with greater ease and facility, and I can also inspect the bowel as the instrument is introduced. Now, the lamp being adjusted, observe what a distinct picture is presented through the speculum: two of the so-called "rectal valves" can be seen very plainly. We will next test their elasticity by means of this hook. Notice how stiff and unyielding they are. I also want you to observe closely the dry and glazed condition of the mucous membrane. There seems to be nothing of special importance in the sigmoid.

Now, one word in reference to the folds of Houston and their relation to constipation or obstipation. A certain amount of resistance in these structures is physiological, and you must acquire a method of distinguishing between that and the pathological resistance. I hardly know how to describe that condition so that you will be able to make the distinction; yet I have found that it is all-important to be able to do so. The rule given us by Martin, that every valve that is not effaced by the hook is diseased and should be divided, is incorrect, according to my observations. After you have examined the "rectal valves" of a series of these cases—those whose bowels are regular and healthy, as well as those who are suffering from obstinate constipation or obstipation—I think you will corroborate what I have just stated. I do not mean by this that these structures are not important factors; far from it. They are the most conspicuous structures in the rectum and are frequently the seat of important rectal pathology. There is no doubt in my mind that they are sometimes the cause of obstinate constipation. Cutting the stiff "valve," however, does not always cure the obstipation; something more is required. I speak now not only of cases which I have operated upon myself, but also of cases that have been operated on by others. A lady from an Ohio town who had had her "valves" cut was sent to me last April by her physician. She still had large obstructive and cicatricial plicæ. The latter condition was due to the cutting. She also had chronic interstitial proctocolitis and severe pain in the right inguinal region. I placed three of my automatic clips on these structures. All of them passed by the end of the second week. I then treated her for the next six weeks with proctocolonic lavage. I received a letter from her a few days ago, in which she stated that her bowels had been perfectly regular since she returned home, moving every morning. This

patient had suffered from obstipation for years: her stools were always hard and lumpy, and it was very difficult to get a movement even with medicine or injections. I have found that in many of these cases presenting the symptom of obstinate constipation there is not a distinct stiffness of the valves, but a marked chronic hypertrophic proctocolitis; the obstruction to the fæces is due to this hypertrophic condition, and not to stiff valves, and I assure you it is as distinctly obstructive. You notice that two of the plicæ in this patient, one on the left side and the other on the right, are distinctly stiff, fibrous, and unyielding, and they obstruct defecation in this case. We shall divide these valves by means of a little instrument which consists of two plates which are connected by means of a spring. With the introducer I now carry the clip through the speculum, gently separate the blades, and drop the clip over the "valve." The introducer is then carefully removed. The clip will pass in from six to twelve days, bringing with it a section of the valve which it will remove by pressure necrosis. I shall then treat the patient twice a week for from four to six weeks with proctocolonic lavage.

The next patient to whom I wish to invite your attention was referred to me by Dr. George F. Hawley. He is fifty-eight years old, and there is a large bluish-red tumor in the left lateral quadrant, just external to the anus. This is what is known as a thrombotic pile. It was my intention, when I first saw him, to remove this tumor at my office and let the patient return home, with the expectation that he would resume work the following day, or the day thereafter at the latest. He was positive that he did not have any other rectal trouble, and I had practically accepted this as true and did not intend making any further examination, when I noticed what appeared to me to be a drop of pus emerging from the anus. I then made a careful examination, and found a small ulcer situated in the posterior quadrant, with a fistulous opening entering it. I also discovered that he had large internal piles, as you can readily see. I then advised him to go to the hospital for this examination and operation. You will observe, as I carefully and thoroughly, though not too greatly, stretch the sphincter and irrigate the rectum, that the field is perfectly clean. That beautiful clean field is obtained by giving the patient the second night before the operation a good cathartic, and following it the next morning with a half-ounce

of Rochelle or Epsom salts; then, about ten o'clock the night before the operation, a colonic flushing is administered, and two hours before the operation, the following morning, an injection of about one-half pint of cool water is given. This method of preparation gives a clean field in which to operate. If you follow the advice contained in most of your text-books—giving a cathartic the previous evening and a high rectal injection about an hour before operating—you will frequently be confronted with a dirty field in which to labor.

You have probably observed that I always operate with the patient in the lithotomy posture and that I sit on a rather low stool. I formerly placed my patient on one or the other side, but it cannot be gainsaid that the lithotomy position offers the best view of the field. Notice that, after the external parts have been made clean with soap, water, alcohol, etc., I irrigate the rectum with a 1 to 3000 bichloride solution and follow this with normal salt solution. I now grasp each anal quadrant at the mucocutaneous junction with a pair of these T-forceps, which are constructed so as to secure the strongest grasp possible with least injury to the tissues. This is very important, especially when operating under infiltration anæsthesia. The forceps being attached, notice what a good command of the field is presented. You see each hemorrhoid just as distinctly as the fingers on one's hand. There is no other method of exposing this field that will give you such a full and complete view. Observe how plainly the ulcer in the posterior quadrant also is exposed. There is a short sinus extending up the bowel beneath the mucous membrane; we shall now carefully lay this open, and dissect out the base of the ulcer. We shall next, with much care and precision, remove an ellipse from each of these hemorrhoidal tumors commensurate with its size. How do I control the hemorrhage? Just as you do when you cut a blood-vessel in doing an operation on any other part of the body. If torsion does not control it, you put a ligature around the vessel. That is exactly what I do, only I have not, as yet, found it necessary to resort to this procedure, and I have probably removed one thousand or more hemorrhoids by this method.

The operation completed, we now dust the field with some antiseptic powder; then pass into the anus, through this speculum, a rubber-covered tampon. The ordinary dressings and a T-bandage

complete the procedure. The dressings will be removed at the end of forty-eight hours, and the patient given a physic. There will be little or no pain during the bowel movement. The parts should be kept clean until healed. He will then return to work in a week or less.

My observations teach me that this is the most rational and radical method we have for operating on this very prevalent malady. No other method will give your patient so little pain, no other will get the patient out of bed in so short a time, and no method is more radical. I insist, however, that the technic must be carefully observed to the letter; otherwise you had better continue to employ some of the other methods,—such as the ligature or the clamp and cautery method,—which not only are painful, but are not in conformity with good surgical principles. It is a physical impossibility for one to do a radical operation on a well-defined case of internal piles and preserve the contour of the anus with either of these methods. It is impossible for any surgeon to grasp a hemorrhoid with a pair of forceps, as is usually done in these operations, and either throw a ligature around it or pull it into a clamp, without removing an excessive amount of the mucous membrane. A hemorrhoid is a bunch of diseased blood-vessels covered with crippled mucous membrane, and in operating you should remove the diseased structures—the blood-vessels—and try to facilitate the reconstruction of the crippled mucosa. To do this necessitates the eversion of the "pile-bearing" area, which is, as you have seen, the first step in our operation; it brings the entire field and all of the piles into plain and distinct view.

The next patient is a married woman, thirty-three years of age. It is through the courtesy of Dr. Worthington, who referred her to me for treatment, that you have the opportunity of witnessing this examination and operation. You will notice in the posterior anal quadrant a crack that extends about one inch in the longitudinal axis of the anus. This is an anal fissure. Observe how firm and fibrous its edges and base seem to be, and that it is triangular in shape on opening the anus. A point to which I wish to call your especial attention is that you do not see here the so-called "sentinel pile." Ball lays much stress on the association of this pile with fissure. He is of the opinion that the majority of fissures are produced by the tearing down, during the passage of a hard motion, of

the lateral attachments of one of the valves of Morgagni. He claims that the remnants of the torn-down valve constitute the "sentinel pile," and that in the thirty cases which he reports it could always be demonstrated; although in some cases it was very small and apparently insignificant, yet in the vast majority it was greatly hypertrophied and œdematous. This ingenious explanation, which is detailed at considerable length in his most excellent treatise on "Diseases of the Rectum and Anus," doubtless is correct in some cases; but that it is the universal cause we cannot admit, and as evidence of our position I ask you to inspect carefully and closely the case which we have before us. You do not see here the slightest indication of a ruptured anal valve. Constipation is regarded as one of the chief causes of fissure ani; but what is the cause of the constipation, or what are the conditions present in constipation? Is it not true that in many cases of constipation and obstipation we have a chronic proctitis, and the relief of the costive condition depends upon the treatment of the proctitis? In some of these patients you also frequently find a dry and inelastic condition of the skin surrounding the anal margin. This proctitis and anitis favor the breaking or cracking of the skin and mucous membrane, which is the initiative in the development of an anal fissure. The cracking may be produced by the passing of large, dry, and hard fecal masses, by the use of improper articles as detergents, and by a lack of cleanliness of these parts. It is astonishing how little hygienic care and attention some people give to the anus and contiguous structures; as Mr. Allingham says, "Most people seem to forget that the anus requires quite as much washing as any other part of the body."

Treatment.—The treatment may be considered from both the palliative and the operative view-point. The palliative treatment is successful in some cases, but, as a rule, I can hardly say it is altogether satisfactory. If there is much pain during defecation, I prefer to move the patient's bowels at night just before retiring. This is best done by an injection. The patient is then more apt to be free from pain during the day. As a local application nitrate of silver has proved the most effective agent in my hands. I use, as a rule, a solution of from one per cent. to two per cent. This is applied with cotton on a probang.

The operative treatment, however, is much more satisfactory.

Divulsion may be accomplished by anæsthetizing the patient, then carefully and thoroughly stretching the sphincter, massaging the fissure, and cutting off the hard edges or tabs of skin that may be present. After the muscles have thus been stretched the contractile power of the sphincter is suspended for a few days, during which the fissure is given an opportunity to heal. In addition to the stretching, an incision may be made through the base of the fissure and all irregularities or tabs of skin cut away. In many of these cases, however, there is no need of giving a general anæsthetic; a one or two per cent. solution of cocaine may be injected beneath the base of the fissure, along its entire length, when it can be painlessly divided and packed with a piece of rubber tissue, then with cotton, and a T-bandage applied. The bowels must be kept loose with some aperient. Should there be much pain, let the patient sit for twenty or thirty minutes in a hot sitz-bath. Should the wound in the skin heal more rapidly than the one on the mucous surface, it may be necessary to divide that part of the tract a second time. We thought it best to give this patient a general anæsthetic because of her nervous condition. Having thoroughly stretched the sphincter and removed the irregularities and the few piles which she had, we will now introduce a rubber-covered tampon and apply the usual dressings.

[Note.—The first case in the above series left the hospital and returned to his work the following day, Wednesday. The second patient left the hospital the following Friday, visited his office, and returned to work the next day, Saturday. The third case was up the following Friday, and returned home on Sunday.—J. R. P.]

TREATMENT OF DILATATION OF THE STOMACH BY GASTRO-ENTEROSTOMY.

CLINICAL LECTURE DELIVERED AT BEAUJON HOSPITAL.

BY G. M. DEBOVE, M.D.,

Dean of the Paris Faculty of Medicine.

Gentlemen,—When I lay before you the history of a patient in these clinical lectures I am usually obliged to rely on the patient himself—on his more or less accurate recollection and his often very uncertain replies—for the details as to the antecedents, beginning, and evolution of the disease which I wish to describe. To-day, however, the conditions are unusually favorable, since I am about to speak of a man whom I have known personally for years, who has often consulted me, and therefore I have been in a position to follow the evolution of a simple ulcer of the stomach that resulted in a stricture of the pylorus necessitating gastro-enterostomy.

D. is a man forty-one years of age, whose antecedents show nothing specially worthy of attention. He says that even in his infancy he manifested a tendency to develop flesh, which you would hardly suspect on seeing how thin he is at present. He has always been a large eater, and has been in the habit of drinking a great amount of liquid; but he was in nowise an alcoholic patient, and confined himself chiefly to wine and water.

When he entered the navy his height was one metre and sixty-nine centimetres and his weight eighty kilogrammes, ten kilogrammes above that normal in men of his height. Under the influence, however, of the physical exercise that the service requires, his weight decreased, and when, after five years, he was discharged, it was but sixty-six kilogrammes.

At the age of twenty-six he married, and all of his misfortunes date from that time.

I often draw your attention to the influence of the mind over the body, and endeavor to convince you that mental trouble has a

deeply disturbing effect on the bodily organs. The stomach appears to be even more susceptible to this influence than the other organs, and it reacts not only by giving rise to the painful complaint known as nervous dyspepsia, but even its secretions may be altered in quality.

With our patient, owing to great grief due to the loss of two children, one of whom died of meningitis at two years of age, and the other at ten months of some unknown complaint, gastric disorder made its appearance, attacks of acidity and vomiting recurring every two or three months. At this time (thirteen years ago) he consulted me, and I made the diagnosis of hyperchlorhydria, and prescribed sodium bicarbonate with chalk. Improvement followed this treatment and lasted for seven years; whenever the slightest gastric symptoms manifested themselves, they were promptly relieved by a short renewal of the treatment.

Three years ago the patient lost weight, under the influence of a long spell of diarrhœa; but he was once more relieved by large doses of prepared chalk, which had the twofold advantage of producing constipation and of counteracting the hyperchlorhydria.

Two years ago his wife died of uræmia during the course of chronic nephritis. From that time his gastric disorder became worse, and he finally showed all the signs of dilatation of the stomach with gastric stasis, and I made the diagnosis of stricture of the pylorus due to cicatrization of a gastric ulcer.

At the beginning I had made the diagnosis of simple hyperchlorhydria; but the unmistakable signs of stenosis of the pylorus obliged me to change my diagnosis to that of simple ulcer.

Simple ulcer is, so to speak, a complication of hyperchlorhydria, from which it can be distinguished only by the occurrence of hemorrhage, hæmatemesis, or melæna. When these symptoms are lacking, hyperchlorhydria and simple ulcer manifest themselves by pain of the same intensity, character, and seat, and occurring at the same time, by similar vomiting and by identical disturbances of the chemistry of the stomach.

The first signs of stenosis of the pylorus appeared, or rather were first noticed, about a year ago. We began to extract by the catheter in the morning, on an empty stomach, alimentary residue, which became more and more abundant; the stomach was manifestly very much dilated. Finally, the patient's general state be-

came so serious that we advised him to enter the hospital, where his condition was found to be as follows:

He had become much thinner, weighing but sixty-one kilogrammes. The disturbance of his organs was limited to the digestive apparatus. His appetite was retained and his thirst was very marked. This retention of appetite is an interesting sign, which in cases where one hesitates between an ulcer and a cancer, leads us to favor the former diagnosis. Gastric pain was no longer so intense as it had been, and no longer recurred so regularly two hours after eating; it was bearable, but continuous, and was not entirely relieved by the administration of alkalies, which in the earlier stages had had such a wonderfully sedative effect.

Vomiting, which only a short time before had been unusual but extremely copious, amounting to several litres at a time, and often containing food that had been taken several days previously, had become much less abundant but of daily occurrence; it really consisted in free regurgitation. The matter vomited never showed any traces of bile, which in this instance is of great importance, since it would be difficult to conceive of bilious vomiting in stenosis of the pylorus, which would naturally prevent a reflux of the contents of the intestine into the stomach.

The vomiting, together with the other symptoms just described, was sufficient to settle the diagnosis; still, it was our duty to resort to the other modes of exploration.

The sound of gastric gurgling was very distinct and could be detected over a large area; though this sign is, in my opinion, of no great value, since in patients whose abdominal wall is very supple it can be produced by simply disturbing the gases of the intestine. The latter sound must not be mistaken for that produced by the movement of liquids in the stomach, which is a mistake liable to be made by a physician not on his guard against this cause of error.

We then distended the stomach to a maximum by administering six grammes of sodium bicarbonate, followed by four grammes of tartaric acid, and in this way convinced ourselves both by inspection and palpation that the stomach was enormous; and, further, that this condition may obtain without the existence of stasis due to pyloric obstruction. In the case of this patient, however, there was a sign which rendered it almost certain that there was obstruction.

By making a series of little knocks on the skin of the abdomen in the region of the stomach distended by gas, the latter organ reacted by a series of contractions that were visible through the wall of the abdomen, and that revealed the efforts it was making to evacuate its contents through a narrowed orifice.

The diagnosis of gastric ectasis due to pyloric stricture was, therefore, probably correct, but it could positively be ascertained only by an examination with the catheter.

In washing the stomach every morning we had drawn out several litres of stagnant liquid presenting the fetid odor and the slightly brown color habitual in such cases. After having carefully cleaned the stomach by a series of washings, we gave a test-meal consisting of two hundred and fifty grammes of distilled water and sixty grammes of stale bread. On the extraction of this meal after an hour's time we found in the liquid thus obtained a considerable amount of free hydrochloric acid,—2.11 per thousand.

The foregoing procedures showed that our patient was suffering from dilatation of the stomach due to an obstacle at the pylorus; but was this obstacle a cancer or a healed ulcer?

I set aside the question of dilatation of the stomach due to pyloric spasm, the existence of which condition has been chiefly affirmed by surgeons who, in operating in certain cases of dilatation, did not find any obstacle at the pylorus. I do not deny that such a spasm may exist; but hardly think that it can give rise to a marked case of dilatation, and I even wonder whether in some cases this spasm has not served as an excuse for surgical operations which, on opening the abdomen, did not appear to be justified.

In the present case ulcer and cancer seemed to me the only causes worthy of discussion, and I decided in favor of an ulcer for the following reasons: the long duration of the disease, and the presence of so large an amount of free hydrochloric acid. Still, these two signs have no absolute value, since an ulcer may degenerate into a cancer, and, on the other hand, I have observed the secretion of hydrochloric acid in cancerous patients.

Our diagnosis of stricture by simple ulcer was afterwards confirmed by laparotomy.

What was the more advisable treatment to prescribe for this patient,—a medical one or a surgical intervention?

The medical treatment consists in removing by the catheter the

substances that accumulate in the stomach, in washing out that organ, and in neutralizing the acids that are secreted or that arise by the different forms of fermentation that take place in it. This is, however, only a palliative treatment; the pain is relieved, but the patient continues to lose strength, partly through absorption of the products of the toxic fermentation that goes on in the stomach, and particularly through lack of proper nourishment. To temporize for any length of time would jeopardize the interests of the patient, who would soon find himself in unfavorable conditions for operation. It is, therefore, necessary in such cases to get the patient to consent to an operation at the earliest practicable moment, as there is no possible chance of spontaneous recovery.

For these reasons we asked Dr. Tuffier to perform gastro-enterostomy. It would be beyond my province to describe this operation in detail; but it was performed with rapidity and skill, and has given good results, which you will presently be able to see for yourselves.

I wish merely to call your attention to two peculiarities noticed during the operation.

The first was that there was no tumor in the region of the pylorus; that part of the stomach was immobilized and adherent to the neighboring organs, and presented manifest signs of inflammation. These observations during an operation have always to be made rapidly, as the patient's welfare requires that scientific curiosity should not be carried too far.

The second point is that the greater curve of the stomach had risen above the umbilicus and that the gastric walls appeared to be very much thickened. The stomach had previously been carefully and completely evacuated, and in such conditions it is not unusual to see the organ, whose muscular tunic has undergone hypertrophy, present only a moderate degree of dilatation at the moment of the operation.

The operation was performed on May 4, and during the first days a rise of temperature gave us some uneasiness. The diet consisted at first of milk, eggs, and soups; but by May 16 the patient was eating anything, and could be considered almost cured.

The benefits derived by this patient from the operation are very great. His appearance is good, he no longer suffers, nor is he taking any medicine. He eats everything without any gastric

disturbance, having no more pain, acidity, vomiting, or eructations. He has gained five pounds in a month, his strength is returning, and everything leads us to hope that he will eventually entirely recover his health.

You will naturally ask, What has become of the functions of the stomach? This organ is still dilated, as we ascertained by again producing artificial distention by the means mentioned above.

Since a new orifice of communication has been made between the stomach and small intestine, it may at first appear strange that gases arising in the stomach do not pass at once into the intestine; experience has shown, however, that in the majority of cases the new orifice is fairly continent. This is due to the fact that the circular muscular fibres of the stomach have alone been cut, the longitudinal fibres having, as it were, simply been separated; in this way the two edges of the orifice remain in contact when gastric tension is only moderate, but the orifice opens to allow the food to pass when contraction of the organ is brought to bear on it.

Still, you must not think that this is always the case, since with another patient, whose history I published in detail, the gases passed immediately from the stomach into the intestine and produced general distention of the abdomen.

But although the stomach of this patient is still dilated, or at any rate easy to distend by alimentary gases, stasis has almost disappeared; when the stomach is washed out in the morning before eating only a few particles of food now come back. I think we must attribute this good result to the fact that this patient's stomach has retained its complete power of contraction, since we saw that during the operation, when it had been emptied, its lower edge rose above the umbilicus.

The hyperchlorhydria still remains and has even increased, and after a test-meal it is now three per thousand; but this appears to do no harm, as the patient has no pain. Furthermore, experience has taught us that this condition steadily decreases, and the reason for this is easy to see. At the beginning, under nervous influence there is gastric hypersecretion, and this acid secretion irritates the mucous membrane, and is at the same time both cause and effect. When the flow into the intestine has lessened the irritation of the mucous membrane, the hyperchlorhydria diminishes.

In closing let me remind you that gastro-enterostomy is not

only a treatment for simple ulcer, but for pyloric stricture from whatever cause.

A few years ago I had in my wards in this hospital a patient suffering from pyloric stricture due to swallowing a large amount of caustic. She was operated on by Dr. Lejars. At the time of the operation she weighed thirty-eight kilogrammes, but after it she gained rapidly until she weighed fifty-one kilogrammes, and her appearance became most satisfactory.

Gastro-enterostomy has frequently been performed for cancer of the stomach. I published the history of such a case in 1895, in which the patient increased thirty-four pounds in three months, and was able to return to work. He afterwards died from complete cachexia due to generalized cancer, but he had no further gastric trouble. In such circumstances the operation is only palliative and prolongs life.

Gastro-enterostomy has also been advised for patients suffering from Reichmann's disease, or continuous secretion of gastric juice; but as a general thing in such circumstances the administration of small and repeated doses of an alkali is enough to improve the situation, without having recourse to a surgical operation.

SURGICAL INTERVENTION IN CASES OF GREAT DILATATION OF THE STOMACH.[1]

CLINICAL LECTURE DELIVERED BEFORE THE MEDICAL DEPARTMENT OF THE MEDICAL FACULTY OF NAPLES, AT THE DEMONSTRATION OF A CASE OF GASTRECTASIS DUE TO HYPERPLASTIC STENOSIS OF THE PYLORUS.

BY PROFESSOR ANTONIO CARDARELLI,

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Gentlemen,—In the case of this woman, who has been examined in your presence, the important fact is the remarkable dilatation of the stomach, in conjunction with considerable lowering of this entire organ,—gastrectasis with gastroptosis. It has been possible to ascertain the degree of dilatation, not only by all the means of physical diagnosis, but also by the insufflation of the stomach (which was performed before you), without distending the gastric walls to an excessive degree. The gastroptosis in this case is no less remarkable. Even without artificial inflation of the stomach, you have been able to note that the gastric region was considerably depressed, and that the lesser curvature of the stomach was defined in the middle of the xipho-umbilical line, while the greater gastric curvature was to be seen at from six to seven centimetres below the umbilicus.

Confronted with such striking forms of gastric dilatation, the clinician's first thought is of pyloric stenosis, as such extensive gastric ectasia cannot easily be produced by those agencies which were once regarded as the causes of the common dilatations of the stomach. Nevertheless, I would not be understood as asserting that this condition of affairs is absolutely impossible, for there may be considerable gastrectasia without a corresponding pyloric stenosis, and, as I shall show later on, depending on quite different conditions.

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[1] Reported and translated by Léon Lebovici, M.D. (Vienna), Carlsbad.

Having, however, admitted the presence of pyloric stenosis in this case, it will not be very difficult for us to determine that which is of the greatest importance,—viz., the nature of the stenosis. Is its character hyperplastic or neoplastic? Has it developed after an inflammatory process in the pylorus, or is it due to a neoplasm of that orifice?

It will be easy to make a correct diagnosis if we study the many and important facts which are furnished by the examination of the patient and the history of the development of her disease. This examination, while showing excessive dilatation and a lowering of the entire stomach, does not reveal any evidence of tumor. There is no resistance and no hardness in the region of the pylorus. There has been no difficulty in discovering the exact condition of the pylorus, because, the patient having lost much flesh, the abdomen was easily accessible to thorough examination. Moreover, the stomach being lowered, we have not been confronted by one of those cases, by no means rare, where the pylorus is concealed behind the liver, and hence inaccessible to observation.

Even without attributing to the fact the importance attached to it by other authors, the presence of hydrochloric acid in the gastric juice may, combined with other signs, be regarded as sufficient indication of the absence of carcinoma. Above all, we must take into consideration the way in which the disease has developed in our patient. It began with daily attacks of pain, of considerable severity and lasting for several hours, combined with acidity and pyrosis. Later these attacks were followed by emesis which supervened from four to five hours or longer after eating. The vomit consisted of acid substances and never contained blood. These facts also point to the presence of gastric ulcer, or at least of inflammatory processes, rather than to carcinoma.

Finally, we must give some consideration to the general condition of the patient. She has lost much flesh, she is quite pale, and her strength is impaired. These changes took place in less than ten months, which fact might point to carcinoma. But we must consider that, concurrent with the primary disease, the patient was subjected to continual vomiting and painful digestion. Thus the impairment of her general health is, independent of the underlying gastric process, due to her sufferings and to the lack of normal assimilation. Moreover, it must be taken into account that the

cachectic condition of the patient is not that of one affected with carcinoma. True, she is badly nourished and pale, but she has not that straw-yellow color seen in cancerous patients and which at once arrests the attention of the practitioner. All these facts, which taken singly would possess but little diagnostic value, are of real importance when considered as a whole.

Our anatomical diagnosis of the case has shown us gastric dilatation and hyperplastic stenosis of the pylorus. In regard to the first we are certain, because the condition was proved by all the methods of physical diagnosis. As to the stenosis its existence is very probable. You will soon see why, setting aside the nature of the stenosis, we can never be entirely certain of its presence, even when we have to deal with excessive dilatation of the stomach, as in this case.

But of what use would this diagnosis be to us, even if we could absolutely determine the character of the process which has produced the pyloric stenosis and thus induced the gastrectasis? If, like the bladder, the stomach were merely a receptacle, we might be satisfied with having recognized its inability to empty itself, owing either to stenosis or to some obstacle in the gastric pylorus. But the stomach is not a receptacle. It is a chemical laboratory, and we must try to discover, with the greatest accuracy, the relation between its inability to evacuate its contents and the changes in its digestive powers. The gastric chemism may become impaired by the influence of the dilatation and stenosis, and this, as a rule, is what occurs. Or, as not infrequently happens, the alteration in the functional and chemical activity of the stomach may become the starting-point for the dilatation, and even for the pyloric stenosis, as I shall demonstrate later on. Thanks to the means now at our disposal, particularly by the examination of the gastric chemism, the functions of the stomach can be determined in all their details, and can be accurately noted day by day, hour by hour. The use of the gastric sound gives us an easy means of determining the mobility, the absorption, the secretion, and the chemical activity present; but it would be of no service if in a case like this we could be satisfied with the comparatively easy diagnosis of gastric dilatation with pyloric stenosis, either of hyperplastic or neoplastic character. What useful indications as to treatment could we derive from such a diagnosis? Besides, you must understand that the

diagnosis of pyloric stenosis, which we make to-day with a reasonable degree of certainty, might be proved erroneous, either at a post-mortem examination or at the surgical operation which may yet be advisable.

The physician who has not well studied the functions of the stomach will be unable to understand how it was possible that there should be no pyloric stenosis in a stomach dilated so extraordinarily and which was unable to evacuate itself. The pathological anatomist will also be obliged to class this case with so many other cases of extreme dilatation of the stomach in which the pyloric stenosis diagnosed by the clinician could not be found. But if the physician had carefully studied the gastric chemism and had noted certain modifications of the pyloric contractions which are produced by the chemical composition of the gastric juice and the gastric contents, he would not be surprised at the error of diagnosis which he had committed and would be able to account for it. This knowledge, which the surgery of the stomach has at least demonstrated to us (if it has not furnished it), can be obtained only by studying gastric dilatations in the manner pursued to-day for all diseases of the stomach, whether we are dealing with disturbances that are merely nervous or functional or with gastropathies that are more or less severe, as in the case before us. Only by such study shall we be convinced of the futility, in many cases of gastric dilatation, of the various methods of medical treatment, of the necessity of resorting to surgical interference, and of the great responsibility we assume in relying too much on medical means and hesitating to avail ourselves of the surgeon's help.

In the case before us, as we have to deal with gastric dilatation produced by pyloric stenosis, it is essential to note the following three facts, viz.: the energy of contraction and tonus of the gastric wall; the gastric chemism; and the retention of the food in the stomach, or, as it was once called, the gastric stagnation.

The energy of contraction, which must not be confounded with gastric mobility, must be determined in order to judge what strength the stomach has conserved to be able to fight against the obstacle in the pylorus. In this case it has been determined with salol and the extraction of the gastric juice from six to seven hours after the regular meal. The degree of the tonus of the gastric wall must be known in every case of gastric dilatation in order that we may

understand what results we have attained by certain therapeutic measures. To a certain extent the severity and frequency of vomiting demonstrate the energy of the gastric contractions; and when, owing either to excessive distention of the gastric fibres or to some other cause, the wall of the stomach is more or less unable to contract, the vomiting may entirely disappear. This last condition might mislead the practitioner to believe that the disease had improved, if it were not for the great stagnation of the gastric contents. The energy of the contractions of the stomach may be estimated by the peristaltic movements discernible behind the abdominal wall, through which, as you have seen in this case, it is easy to distinguish the form of the stomach and its peristaltic and antiperistaltic action. From the more or less rapid and energetic succession of these movements, especially during digestion or under massage of the abdominal wall, we are able to determine the amount of contraction of the gastric muscular fibres. When the peristaltic action becomes very slow or entirely ceases, the cessation being accompanied by stagnation of the gastric contents, we may believe in the presence of paralysis of the wall of the stomach. The energy of the contractions of the gastric muscles may also be determined by the intensity, frequency, and duration of the severe pain which appears in the form of a true gastric colic. But in relation to this also I must repeat what has been previously said in regard to vomiting and the peristaltic action,—viz., that the diminution of the distressing symptoms does not indicate an improvement in the patient so long as the physician recognizes a progressive stagnation of the contents of the stomach.

The tonus of the gastric wall, which is usually in proportion to its energy of contraction, may be determined by such complicated methods as those advocated by Rosenbach and Dehio, but of which I shall not speak at present. The simplest method is to have the patient stand upright, and then find the lowest point of gastric percussive dulness, which in a case of great dilatation with stagnation can be accurately discovered. When the lower portion of the stomach has been clearly defined, the patient drinks a certain quantity of water. Should the gastric wall have lost its tonus the weight of the fluid will lower the level of percussive dulness, whereas if the tonus is unimpaired the limit will not be altered. I have found this method of examination very useful, as also that of

noting the change of percussive dulness with the patient in a lateral position (especially in relation to the bottom of the stomach).

I believe that insufflation of the stomach is useful in judging of the tonus of the gastric wall, and also, to a certain extent, of the energy of the muscular fibres. The insufflation of the stomach enables us to see to what degree the gastric wall is yielding, and also the way in which the stomach throws off the gas with which it has been filled,—viz., through the pylorus if that be permeable, or through the cardia if there be pyloric stenosis. I wish to direct your attention to these simple methods because those advocated by Rosenbach and Dehio are much more difficult of application. If it is desirable to avoid the introduction of the sound, distention of the stomach may be induced by effervescent powders. The methods of Rosenbach and Dehio can no longer be regarded as sufficiently exact to determine the tonus of the gastric wall. We cannot tell to what degree the lowering of the liquid in the stomach (combined with the liquid which the patient has been ordered to drink) may be due to the yielding of the gastric wall which has lost its physiological tonus, and to what extent it has been made to depend on a total lowering of the stomach, the latter frequently occurring in cases of gastric dilatation.

The other examination which must be made in every case of dilatation of the stomach refers to the gastric chemism. I intend to touch only upon the digestive activity exerted upon the food by the gastric juice, but if there be great gastrectasia, as in this case, I must insist upon a chemical analysis of the gastric juice and the contents of the stomach. This analysis not only elucidates the diagnosis but also furnishes useful therapeutic indications. The examinations bearing upon the total acidity of the contents of the stomach in general and on the hydrochloric acid in particular are most important. As before said, the presence of hydrochloric acid in the stomach may be regarded, at least in conjunction with other symptoms, as excluding carcinomatous origin for the pyloric stenosis. And if we see such excessive dilatation as in the present instance, it is always of the utmost importance to be able to remove all doubt as to the presence of a neoplasm. Moreover, if we can prove the presence of a hyperchlorhydria which is more or less permanent and has attained a comparatively high degree, the fact acquires great importance in the case of gastric dilatation from

another point of view,—viz., an excessive dilatation of the stomach with all its accompanying conditions may arise only from hyperchlorhydria, which renders it unnecessary to search for more remote causes. Under certain conditions of hyperæsthesia of the gastric mucous membrane, or if there be superficial catarrhal erosions of the pyloric region (erosions which do not deserve the name of ulcerations and which leave no traces), the hyperchlorhydria itself is sufficient to produce a spastic stenosis of the pylorus. This stenosis, which during the life of the patient may exactly simulate an organic stenosis, produces dilatation of the stomach. The clinician must always remember this possibility. The thought of a spastic stenosis is based upon the combination of hyperchlorhydria with dilatation of the stomach and a pyloric stenosis, which, though not of an organic character and not to be found either on the cadaver or upon the operated subject, is nevertheless no less active than the organic stenosis. It was to this condition I alluded when I said that it was impossible to make an infallible diagnosis of organic stenosis of the pylorus unless proper consideration was given to the causes which produced it.

In the case of gastrectasis the physician must first ascertain the degree of retention or the stagnation of the contents of the stomach. The latter shows the greater or less severity of the gastric dilatation which must serve as the guide and the rule for treatment; and it is through the stagnation that the physician is convinced of the want of efficiency in his therapeutic measures. I will go even further and say that the dilatation of the stomach, even if it be excessive, if not accompanied by a more or less protracted or a permanent stagnation, would not be an evidence of real disease. Excessive dilatations of the stomach, occupying the greater portion of that organ, are found *post mortem* in individuals who had no gastric lesions and in whom the only evidence of disease was great voracity. Such cases are now and then registered as anatomical curiosities, and they illustrate how little importance may be attached to dilatation of the stomach without accompanying stagnation of food.

Early in my experience in the "Ospedale degli Incurabile" I performed a post-mortem examination of a woman who had had pylosarcosis and diabetes. She had been an object of curiosity owing to her enormous appetite. She ate eighteen cutlets and

forty eggs in one day. Her stomach depended like an apron over the entire anterior surface of the abdomen from the epigastrium to the pubic region and was almost empty.

I think that the classification of the dilatations of the stomach should depend upon the greater or less duration of the stagnation and not upon the breadth of that organ. It is the stagnation which produces the severest symptoms that accompany gastric dilatation. The fermentation produced by the food remaining undigested alters the gastric chemism and becomes the source of severe and even serious troubles, owing to autointoxication. The gastric dilatation is increased by the gas resulting from fermentation rather than by the mass of undigested matter itself. The hyperplastic pyloric stenosis and the pyloric spasm are caused by the retention of the contents of the stomach; and even the gastrosuccorrhœa itself, the permanent hyperchlorhydria, becomes aggravated by the stagnation of the food.

The stagnation is of the utmost importance in cases of gastric dilatation and pyloric stenosis, no matter from what cause they may arise. Whether dealing with neoplasm or with cicatricial shrinking, with pyloritis or peripyloritis, or even with simple pyloric spasm, the importance remains the same, as this condition will be found in any of the foregoing diseases. In the case of neoplasm death is mainly due to stagnation, the new growth itself being a minor cause, the stagnation materially hastening the end. The truth of this is proved by the fact that if by means of surgical intervention a free passage is made for the stagnant gastric contents, the first results are extremely satisfactory, even in cases of carcinomatous stenosis. For a time, in fact, the improvement is so great as almost to lead one to believe in ultimate recovery. This fact should be clearly established in your minds, for if, owing to error of diagnosis, surgical interference has been advised, believing the patient to be suffering from simple stenosis, and during the operation a neoplasm should be discovered, rest assured that the patient not only has received no injury but will be materially relieved and frequently much benefited.

It is from the surgery of the stomach that so much information has been derived, the most important being that which refers to stagnation of its contents. Professor Carle, of Turin, whose operations in gastric surgery will remain a monument to Italy and

to surgical science, has arrived at the conclusion that an incontinent stomach is much better than one in which stagnation is possible, producing abnormal fermentative processes. Doyen takes the same ground.

If we apply the foregoing to our special case, we may note the following. The contracting energy of the muscular fibres is considerably impaired, because the vomiting has become less frequent, whereas the food stagnation is permanent, even after long fasting. We cannot attribute this diminution of vomiting to the regular diet to which the patient has been submitted since entering our clinic, because that could be but a partial cause. The pains of gastric colic have materially diminished until they are comparatively insignificant. The visible peristaltic and antiperistaltic movements of the stomach are slow and feeble and occur at rather long intervals. The gastric chemism is rather conserved. But what must be specially taken into consideration is the fact that the stagnation of the contents of the stomach is permanent and considerable, and that medicinal means are inefficacious to remedy the condition. You have noted in this patient the presence of the great stagnation of a moderate quantity of milk ingested more than twelve hours previously.

We will now consider all the so-called medical means available for the treatment of several similar cases, determining at the same time what degree of confidence we may place in those means.

Above all, a strict dietetic regimen must be observed, even going so far as to allow nothing but milk in small quantities, and then only ass's milk. In some instances, as in the present, improvement in the patient may be seen as soon as this regimen is adopted; in others, despite the rigid regimen, the vomiting and the gastric colic continue. Leaving this fact aside, we find that the gastric stagnation remains; and, moreover, many patients cannot bear the milk diet, which seems only to increase the fermentation.

To the strict dietetic regimen are added those remedies and means which are supposed to increase the tonus and the energy of contraction of the gastric wall. Ergot and the preparations of strychnine are the chief remedies used, in connection with the application of cold, the epigastric douche, massage, electrotherapy in all its branches, and the girdle of Glénard.

Now, apart from the fact that such remedies as ergot and strych-

nine are useless in most cases, even in those of dilatation of the stomach due to paresis of the gastric muscles, of what possible service can they or any other of the means mentioned be in cases where there is gastrectasis owing to pyloric stenosis? May we hope to remove the obstacle by giving the gastric wall greater power of contraction? Do we not see, by means of the peristaltic and antiperistaltic movements of the stomach, that the gastric wall is already doing its utmost to repel the mass? And with our therapeutic means what more could we do than the muscular fibres have already done before becoming affected with the flaccidity which we are trying to overcome?

I by no means wish to be regarded as undervaluing the importance of therapeutic measures, particularly in the case of dilatation of the stomach and gastric stagnation. I only desire that the use of the means shall be confined to those cases in which the gastric stagnation and dilatation have not become extreme. Especially would I have it avoided where there is absolute inability of the stomach to empty itself. In such cases the use of the sound and the washing out of the stomach, though they may relieve the patient, are not only useless but are liable to damage the organ itself.

In all this I have, as before said, no desire to create in your minds a scepticism towards the efficacy of medical methods, but rather to inspire in you an increased confidence in the use of surgery. I will go even further and declare that a physician of to-day takes upon himself a great responsibility when he persists in the use of medical means beyond certain limits in treating cases of pyloric stenosis. If surgical intervention is not resorted to until too late, the practitioner is deserving of severe criticism.

In regard to this unwarranted delay, Dr. Fantino, in his remarkable work published three years ago, in which are reported the splendid results obtained by Professor Carle, of Turin, deplores the fact that so many cases of supposed simple stenosis or other non-malignant processes of the stomach are presented after the patient has been physically reduced to the last extent and has suffered years of pain. He says, and quite justly in my estimation, "This evidently results from the fact that many physicians are not yet impressed with the inefficacy of certain medical means and of the relative value of surgical intervention if resorted to in time."

Professor Doyen contends that all the severe dyspepsias which do not depend upon carcinoma and in which medical means have been powerless belong properly in the domain of surgery. This conclusion of Doyen may contain some exaggeration, but if one believes that all those cases of gastrectasis which have obstinately resisted the physician's efforts to remove the gastric stagnation should be put in the surgeon's hands, the statement of Professor Doyen is clearly true. And if one is convinced that the dilatation is due to pyloric stenosis and that this is not of neoplastic character, failure to invoke the surgeon's aid means, on the part of the physician, a great sin of omission.

If you ask me to what extent we may confide in medical treatment, I would reply without hesitation that the moment you have certainly diagnosed simple pyloric stenosis, with more or less permanent stagnation of the gastric contents, you must resort to surgical interference. I go further and advise surgery even if you are not entirely convinced of stenosis, but have discovered the absolute inability of the stomach to evacuate its contents and a state of permanent stagnation. The intervention of surgery will be all the more urgent if there is also found dilatation of the stomach to such an extent that it seems impossible that the organ should ever return to its normal size or that it could evacuate itself in its present shape and abnormal position (as, for instance, in the case before us). The necessity for operation may again be indicated by the quality and quantity of the gastric contents extracted by means of the sound. The quality and quantity of the urine and the rate at which flesh is lost should demand equal attention. Also should be taken into consideration the miserable general condition of the patient, arising from protracted suffering and a life of privation. Under such conditions, when surgery has been known to give back to patients reduced in the extreme, not only life but absolute health, how can the physician hesitate for a moment as to the use of surgical intervention?

In all frankness, I believe the reason to be in many cases because the practitioner is not altogether certain of his own diagnosis, and he fears that the surgeon's knife will reveal his error. To remove this fear, it should be understood that mistakes in this kind of diagnosis have been made by the most prominent clinicians in such cases, even after long and careful observation of the pa-

tient. Besides this is the fact that error of diagnosis does not lessen the benefit and relief derived from surgical intervention.

Into what errors of diagnosis may one fall? One may, *e.g.*, diagnose a stenosis due to cicatrices following ulceration, and instead thereof find a pyloritis or a peripyloritis, without any trace of ulceration. Or, again, a hyperplastic process in the pylorus may be diagnosed, when peritoneal adhesions were the cause of the stenosis, the pylorus being healthy. Again, the presence of a hyperplastic stenosis has been determined, and there has been revealed instead a circumscribed and annular neoplasm. Lastly, as the greatest diagnostic error on the part of the physician, the surgeon may find that the pylorus is quite free from any affection, and that the excessive dilatation and stagnation arise from a permanent hyperchlorhydria, with a relapsing or a more or less lasting spasm of the stomach. All the foregoing errors of diagnosis have been committed. Nevertheless, the results of the operations have been beneficial, because surgical intervention removed the stagnation, independently of the cause which produced it.

In order that you may thoroughly appreciate the practical considerations which we have discussed, I will mention some new facts clearly demonstrated by gastric surgery. Surgery of the stomach has revealed to us the frequency of pyloric spasm and of pyloritis and peripyloritis due to simple functional changes or to changes in the gastric chemism. To this same surgery is due our knowledge that, when a free passage is made for the contents of the stomach and gastric stagnation is removed, the dilatation of the stomach begins to diminish; and, what is still more remarkable, the gastric chemism becomes modified, and, if there has been permanent hyperchlorhydria, it decreases and finally disappears. In addition to these results, it is noticed that in a few days the appetite returns and the quantity and quality of the urine become normal, so that nutrition is re-established and a complete recovery follows. This result, excepting only in case of carcinoma, can be obtained in all cases of dilatation of the stomach. There will, however, be some slight differences due to the difficulties which the surgeon may have to encounter as to the technic of operation.

Such statistics as those presented by Professor Carle, which show only two unsuccessful operations out of twenty-nine performed for benign stenosis, fully justify our confidence in gastric

surgery. Setting aside the information I have acquired from books of recent years, more particularly that by Carle and Fantino, I have a personal knowledge of the cases presented in this clinic and which have been operated upon by the able Dr. d'Antona. Of these I wish to put before you the most salient points, in accordance with Dr. d'Antona's own address before the Royal Medico-Surgical Academy of Naples.

I. *Gastrectasis due to Cicatricial Pyloric Stenosis produced by Ulceration.*—A man thirty years old suffered every day from the summer of 1897 with severe pains in the stomach, setting in two hours after each meal and disappearing with vomiting. In the material thrown off there were sometimes streaks of blood. The patient showed, besides his cachectic condition, extreme dilatation of the stomach, active peristaltic and antiperistaltic movements, and signs of stagnation. Examination of the contents of the stomach showed the absence of free hydrochloric acid and the presence of lactic and butyric acids. On April 20, 1898, an operation was performed. In the pyloric antrum was found a large cicatrix which also extended to the small curvature of the stomach. The pylorus was so narrow that it would hardly admit the handle of a penholder. The improvement following operation was so steady that the patient was able to take solid food upon the eighth day. Within twenty-eight days his weight had increased by four and a half kilogrammes. He is now absolutely well and has grown very stout.

II. *Gastrectasis owing to Cicatricial Pyloric Stenosis; Disseminated Ulcerations.*—A man thirty-six years old suffered for five years with pyrosis after the ingestion of food. On June 9, 1898, he was seized with excruciating pain in the right hypochondriac region. This was followed some hours after by the elimination of fecal matter mixed with blood. From that time he suffered constantly with pyrosis and pain during digestion, which, however, diminished after the patient was put upon a milk diet and the use of alkaline remedies. On August 20 he vomited from three to four litres of a liquid as dark as coffee-grounds, and fifteen minutes later he discharged through the rectum about a half-litre of red blood mixed with dark fecal matter. From that time his condition became considerably worse. Examination showed the presence of very pronounced *gargouillement,* even when the patient had taken no food. The lower border of the stomach was somewhat below

the transverse umbilical line, and when the stomach was emptied, from seven to eight hours after the usual meal, the food in it was almost undigested. The gastric juice extracted after the standard meal showed an increase of total acidity, a marked evidence of reaction of free hydrochloric acid, and the absence of lactic acid. At the operation cicatricial pyloric stenosis was revealed with small ulcers disseminated over the mucous membrane of the stomach. The patient died a few days later from ulceration due to a too-tight suture around Murphy's button.

III. *Gastrectasis owing to Cicatricial Pyloric Stenosis, with Peripyloritis.*—A woman fifty-one years old suffered for nearly seven years from gastric trouble and pain which sometimes supervened from ten to twelve hours after meals. Vomiting and hæmatemesis occurred frequently, melæna was often present and occasionally there was a copious discharge of red blood per rectum. The stomach was enormously dilated and prolapsed, the small curvature being three finger-breadths above the umbilicus and the large curvature almost reaching the pelvic region. A slight resistance could be felt over the pylorus. There was great peristaltic action, and considerable *gargouillement*. On emptying the stomach from ten to twelve hours after the ingestion of food, a large quantity of undigested material was found. In the gastric juice there was no lactic acid and the acidity was normal. On May 1, 1899, the patient was subjected to gastrojejunostomy, and there was found a cicatricial pyloric stenosis with considerable peripyloritis. Complete recovery followed the operation.

IV. *Gastrectasis owing to Cicatricial Pyloric Stenosis.*—A man forty years of age was three years ago suddenly seized with severe visceral pains, vomiting, and diarrhœa. From this time his digestion steadily became more and more difficult and painful. During the past year copious emesis has taken place every four or five days. In the vomit were found remnants of food taken several days before, but never any blood or bile. He has constantly lost flesh, the stomach has been lowered and dilated as in the last case, and great *gargouillement* was present. There was no tumor and no infiltration. In the gastric juice there was free hydrochloric acid, and no lactic acid could be detected even by the method of Boas. Upon operation cicatricial pyloric stenosis was discovered. His health is now excellent, his weight having increased ten kilogrammes.

CLUB-FOOT; ASTRAGALECTOMY FOR RELAPSED AND INVETERATE CASES.

A CLINICAL LECTURE DELIVERED AT THE UNIVERSITY HOSPITAL.

BY DE FOREST WILLARD, M.D.,

Clinical Professor of Orthopædic Surgery, University of Pennsylvania; Surgeon, Presbyterian Hospital, Philadelphia.

GENTLEMEN,—I present to you to-day two cases of talipes equinovarus of especially severe grade. The one is a neglected and untreated boy of ten. As you will observe by examination of his foot and from this photograph (Fig. 1) he has, by constant walking, forced his astragalus forward, and so distorted its shape together with that of all his tarsal bones that it is impossible again (as the accompanying skiagraph shows, Fig. 2) to replace this astragalus within the inter-malleolar mortise. The other is a rigid relapsed case which from neglect of manipulation after previous tenotomies and forcible straightening, has again assumed its deformed position, and is steadily growing worse, since each step tends to render all the tarsal bones wedge-shaped and distorted (Figs. 3 and 4).

You have in this clinic so frequently seen the benefit of multiple tenotomies and forcible straightening that I need not say that it is the operation of preference; but every surgeon will meet with a class of cases in which the hinderance to correction lies not in tendons and fascia alone, but also in the bones themselves, and it is of these severe grades only that I shall speak this morning while removing the astragali of these two cases.

Astragalectomy is to be reserved for: (1) Adults with great bony deformity; (2) neglected children of five to fifteen years, who have markedly distorted their tarsi by locomotion; (3) relapsed cases which have resisted the milder forms of operation, or which have been neglected by parents after previous operation; (4) only occasionally, young children in whom from infancy the bones of the foot have been exceedingly rigid and unyielding, and where there

is practically but little motion either at the ankle-joint or in the tarsus. Aside from these varieties, practically all cases can be brought into proper walking position by multiple subcutaneous tenotomies, fasciotomies, and forcible straightening, or by open section of the contracted tissues. Let it be understood that any operation for club-foot should be considered incomplete in which at the time of operation the foot cannot by slight pressure be brought into nearly a normal position, and the foot extended to at least a right angle with the leg. The object of every operation for the relief of talipes should be to place the foot in such position that locomotion shall be secured after the lapse of a few weeks, with the sole of the foot squarely upon the ground and with hyperextension at the ankle-joint, which will enable the patient to walk in the normal position. Unfortunately, other conditions besides those of the foot will modify locomotion; the in-toeing which is so common in these cases is not due alone to the twisting of the foot, but is occasioned also by rotation of the legs upon the thighs, and of the thighs upon the pelvis, the external rotators of the trochanters often being defective.

Astragalectomy is an operation that would never be required if the family practitioner could be brought to the comprehension that the treatment of club-foot should be commenced before the infant is twenty-four hours old,—almost, as has been remarked by one individual, "during the third stage of labor." It seems strange that any educated physician can wilfully neglect the golden opportunity of moulding such distorted bones during the first three months of life when the osseous structures are soft, and when growth is so rapid that if neglected, irreparable deformity of the bony structures must result. During these months constant manipulation and simple forms of retentive apparatus will accomplish wonders, but all of these milder methods we will discuss at another time. With the definite understanding, then, that bone operations are necessary only in the class of cases already mentioned, I may say that from my experience, after testing all the bony substitutes, astragalectomy is the simplest and the safest operation, and is the one that yields subsequently the most elastic foot. No cancellated bony structure is exposed, the cartilage-covered calcaneum slips easily into the mortise between the malleoli of the tibia and fibula, and excellent rocking motion is secured at the ankle-joint. When

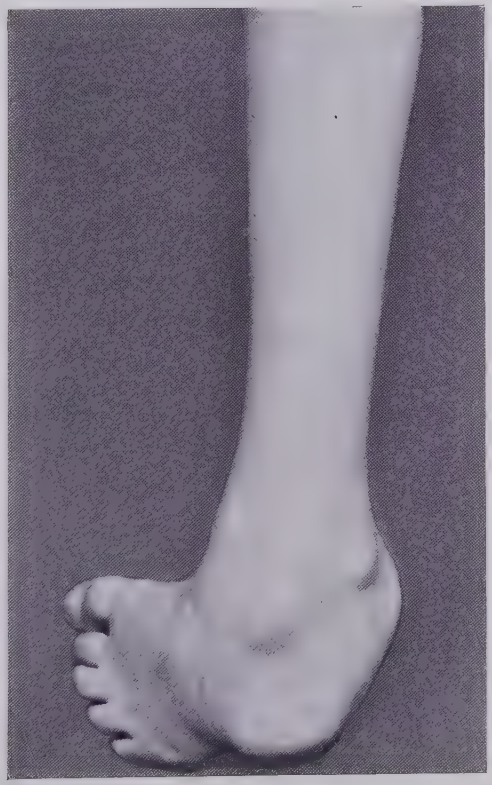

FIG. 1.—Neglected talipes equinovarus; astragalus forced forward.

FIG. 2.—Skiagraph showing a distorted astragalus and the tarsal bones misshapen.

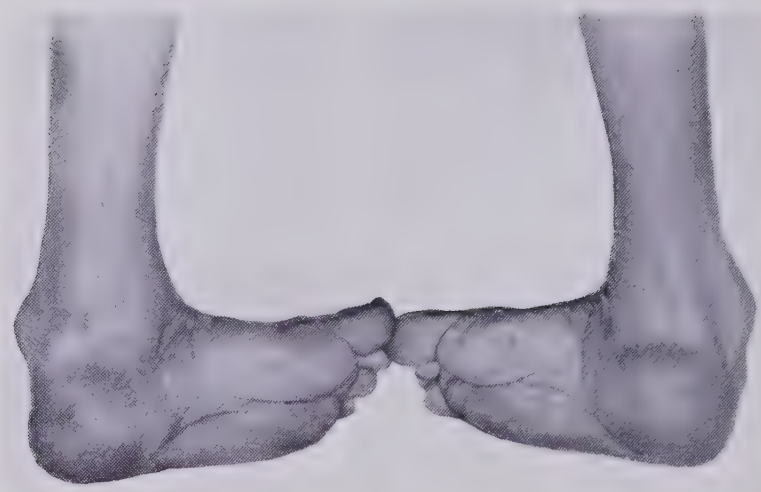

FIG. 3.—Relapsed talipes equinovarus.

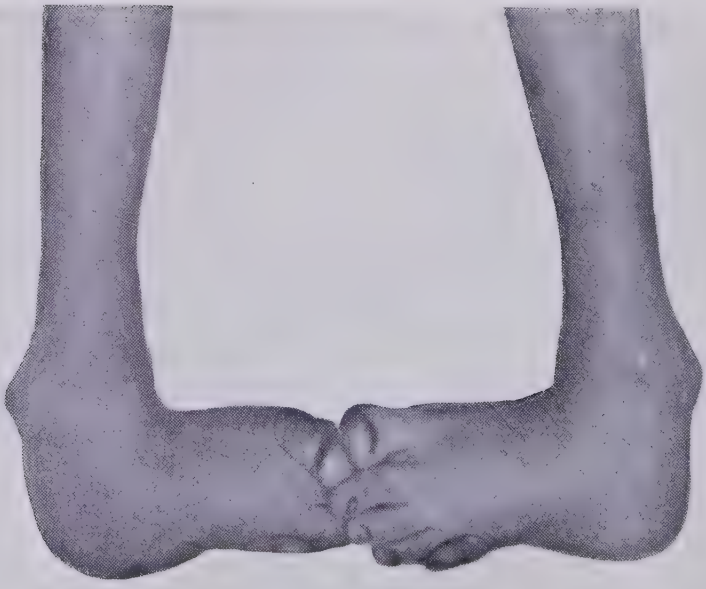

FIG. 4.—Relapsed talipes equinovarus.

both feet are operated on, the slight shortening in stature is unnoticed. The removal of the astragalus alone frequently gives hyperextension beyond the right angle, without any division of the tendo Achillis; room is also afforded for correction of the varus without section of the tibialis posticus and anticus. In my judgment, removal of the astragalus is a decidedly better method than wedge-shaped tarsal excision, or osteotomy of the neck of the bone, or any of the various methods practised. The secret of securing a movable ankle-joint lies in the performing of a thoroughly aseptic section, so that speedy union may be secured, and ankylosis avoided. To prepare for such an operation in an adult with large callosities and bursæ upon the outer side of the foot is a work of days of preparation, lest infection take place. All callosities, corns, etc., must be thoroughly removed by cutting and scraping and the use of washing soda. An excellent plan is to poultice with flaxseed meal for several nights, to soften the horny epidermis; the scraping must then be most vigorous, and the antiseptics made to thoroughly penetrate the skin by wet sublimated gauze applied for several nights, when possible. Rapid healing is so dependent upon cleanliness that time is gained, not lost, by preliminary treatment.

The incision for the removal of the astragalus (as you will note on this boy's foot) commences in front of and a little below the external malleolus, so that the tender scar tissue shall not later be over the tip of the malleolus. The peroneal tendon should be avoided, and as the extensor tendons of the toes are displaced inward, the head of the astragalus can be reached by an incision stopping at the astragalo-scaphoid articulation, just short of these tendons. The distortion of the astragalus is sometimes so great that the error is made of cutting in front of the scaphoid instead of behind it, a mistake which will be recognized by the fact that while both articulations are cup-shaped, in the one case there will be a single bone in front, while in the other there will be three bony facets. By keeping close to the periosteum of the bone upon the dorsum of the foot, wounding of the anterior tibial artery can always be avoided, and the tendons spared. The deformation of the astragalus is often so great that disarticulation becomes difficult, but simple nicks with a knife or scissors will free the bone upon its dorsal and external aspects, after which the internal lateral ligament of the ankle can be reached. When the astragalo-calcaneal ligaments have been

divided, the bone should be seized with a pair of strong forceps by means of which enucleation can be more readily accomplished, especially if an assistant forcibly bends the anterior part of the foot in various directions, as needed. When the bone is soft, care must be taken that it be not crushed in the jaws of the forceps. The twisting of the bone is sometimes so great that after removal it has been unrecognizable even by such a skilled anatomist as Joseph Leidy. When the internal lateral ligament is reached, great care should be exercised lest the posterior tibial artery be wounded. In a few cases of severe grade if the calcaneum is too wide to slip into the mortise of the tibia and fibula, the tip of the latter bone may be cut away, and in exceedingly rare cases a portion of the cuboid may also demand removal.

The Esmarch bandage has such positive disadvantages that I prefer to work without its application, save in exceptional cases. It has sometimes unexpectedly been the cause of paralysis, and nearly always the subsequent oozing interferes with the speedy closure of the wound.

I have already said that the secret of successful treatment depends upon the restoration of the foot to its normal position. Only rarely the tendo Achillis, or the structures upon the side of the foot, will require attention. If any pieces of partially detached cartilage are found in the cavern, they should be dissected out lest they necrose. The wound should then be wiped dry, the skin closed with catgut sutures, and if thorough asepsis has been enforced, a few strands of sterile catgut only are inserted at the angle. If the asepsis has been complete, the surgeon can then encase the foot in gypsum, fix it in proper position, and let it alone for four weeks. A zinc strip should be inserted, and the cast cut throughout its entire length, save for a half inch at the top and bottom as soon as it has hardened. This will save much time, and will also allow for springing open the cast at any time should swelling necessitate, or circulation be impeded. If the toes remain exposed, the condition of the foot can be accurately gauged. At the end of the third week the patient should walk about upon the cast. At the end of the fourth week the cast should be removed, and a proper curative walking apparatus applied so that good ankle motion may be secured. The administration of ether and a little forcible motion at the joint will save the patient pain, and assist in more rapid

FIG. 5.—Talipes equinovarus; result after astragalectomy.

FIG. 6.—Right talipes equinovarus; result after astragalectomy.

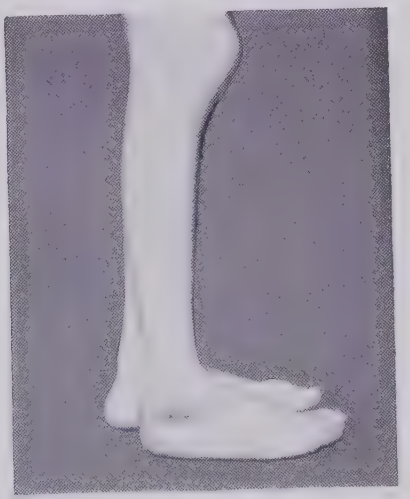

FIG. 7.—Talipes equinovarus; result after astragalectomy.

restoration of function. Manipulation, massage, and voluntary muscular movements should be maintained for many months. In many cases the use of apparatus will be dispensed with in a few months, but not until the tendency to relapse has passed. Too great haste should never be the rule. If you have followed the steps of the operation as demonstrated in these two cases during this hour, you will have fixed in your minds a procedure that will relieve you from the discomfiture of failure in these most troublesome relapsed and rigid cases. A few photographs which I exhibit to illustrate the results of this operation will demonstrate the changes that take place in the appearance of the foot (Figs. 5, 6, and 7), but the best demonstration of its utility will be to show you the walking powers of a few patients upon whom the operation has been performed in the past. They walk squarely upon the soles of their feet, they have good motion at the joints, marked strength in their legs, and a number of them have for years dispensed with all apparatus.

Obstetrics and Gynæcology

ABDOMINAL TUMOR.

CLINICAL LECTURE DELIVERED AT THE POLYCLINIC HOSPITAL, PHILADELPHIA.

BY J. MONTGOMERY BALDY, M.D.,
Of Philadelphia, Pa.

Gentlemen,—To determine the nature of any abdominal enlargement in a woman there are always three things to be considered: is the abdomen merely distended with flatulence, is the patient pregnant, or is there a tumor,—*i.e.*, a new growth? Many a surgeon has opened the abdomen only to find that the swelling was due entirely to flatulence. Of course this has generally been the result of grave carelessness. If the belly be percussed, it will if distended with flatulence always be tympanitic. An enlarged abdomen which is tympanitic all over is sure to be free from a neoplasm, by which we mean a growth sufficiently large to distend the abdominal walls; this of course excludes all pelvic growths from consideration. Tympany indicates that the intestines are directly in contact with the abdominal walls. The only possible chance of mistake in this matter is either where there is free fluid in the abdominal cavity, in which case the intestines are apt to be floated upon the fluid and to lie in close apposition to the walls, or when in making pressure in percussing the walls are depressed to the level of the intestines. In such a case, however, the free fluid will more or less distend the flanks, at which points the percussion-note will be dull; so the test "tympanitic all over" is not present. By simple percussion, therefore, any one of even ordinary skill can insure himself against such a gross error as mistaking an abdomen distended by gas for one distended by a tumor.

Now you will note, as I percuss the patient before us, that the

note is dull over the entire top of the distention. That in itself indicates that in this patient there is something comparatively solid between the abdominal walls and the intestines, and the question of tympanitic distention alone is thus disposed of in an instant.

The second point which arises in the diagnosis is that of possible pregnancy. After settling the question of tympanitic distention, that of pregnancy is of prime importance, and in no case should the physician decide that there is a pathological tumor until he can say definitely, "This is not pregnancy." This point comes first, and should ever remain in mind as a possibility. In the majority of instances the differentiation is a matter of great ease, but there occasionally occurs a case in which the decision is exceedingly difficult. If, after having exhausted all your resources of diagnosis, you are unable to decide definitely, do not make your decision by guess; you have an even chance of winning in a guess, of course, but if you fail, the result may be disastrous to your patient and to your own reputation. In every such case, give the pregnancy the benefit of the doubt; admit frankly that you are unable to decide positively, and advise delay and a future examination. A month or two will surely tell the truth; and even if you are compelled to wait six months, in the vast majority of cases you will have done no harm to your patient, and will have established a certainty. There is one exception to this,—cancer. Fortunately, however, it is not in the cancer cases that the doubt is likely to arise; I never have seen such a case in my experience.

In the case before us it is easy to exclude pregnancy; there are no gastric symptoms, no breast-symptoms, no cessation of menstruation, no discoloration of the vagina. The enlargement, it is true, is about the size of a full-term pregnancy, but the outlines of the distended abdomen are evenly distributed in all directions, and the patient has been aware of a swelling from its beginning, about eighteen months ago. Our digital examination of the pelvis shows a somewhat enlarged uterus, possibly three inches in depth, lying anterior, movable, and evidently not a part of the large tumor; we have, therefore, no trouble in excluding pregnancy in this case. Moreover, allow me in passing to direct your attention to the fact that the digital examination we have just made has eliminated from consideration a large class of pathological possibilities. You note that the uterus is small and is free from the tumor,—is not

a part of it. That means, of course, that the tumor does not grow from the uterus, and that we have at once excluded from the diagnosis all uterine growths, benign as well as malignant. In considering a given case, always try to isolate the uterus; if you can do so, you at once eliminate all uterine growths from consideration.

You may exclaim at my expression, "Try to isolate the uterus," but in many cases you will find this easier to do theoretically than practically. When the pelvis and abdomen are filled up with a tumor or tumors, things are at times so distorted from their natural position, and the examination is so hampered by the distention and distortion, that it tries one's greatest skill to say, "This is uterus and this is not." For instance, two nodules of the tumor may so surround the fundus as to enclose it between them, making impossible its examination from either the back or the front; a tumor may be so placed in relation to the uterus, and so adherent to it, as to render it extremely difficult to say what is what; or, a growth may crowd the fundus backward or forward, or to the side, and the consistency of the growth may be so like that of the uterus that one is puzzled to say definitely whether or not what he feels is fundus or a nodule of the tumor, especially if there be two or three points, any of which could be uterus. And so the difficulties multiply.

Fortunately, the decision is easy in this case, and we have now reached a point in our diagnosis where there is little more to add. If this distention is neither flatulence, pregnancy, nor a uterine growth, what can it be? First, the patient and her friends noticed the enlargement begin close to the pubis, to the right side, and gradually enlarge upward, which indicates that it came from the pelvis. Furthermore, the digital examination through the vagina discloses the fact that the pelvis is encroached upon by the tumor as we should expect it to be if, for instance, the growth was an ovarian cyst. This gives enough support to the supposition of a pelvic origin for us to assume it to be such in our further consideration. Again, all the excretions have been examined, and kidney, liver, pancreatic, or splenic disease has been eliminated; moreover, the growth began at too low a point for a tumor of any of these organs.

The woman's history is as follows: Twenty-seven years old. She had a baby six years ago. Has enjoyed good health until within eighteen months, when the growth was first noticed, and she began to bleed irregularly and profusely; for the past six months

the bleeding has been fairly constant. Now, this is not an ordinary history of uncomplicated ovarian tumors; yet the ovaries and broad ligaments are the only organs left in the pelvis from which such a large circumscribed tumor could grow. The distention is as great as a full-term pregnancy; it is uniformly regular in all its outlines, and there is no bulging in the flanks, such as would occur if there were free fluid in the abdominal cavity; the percussion-note in the flanks is resonant, showing the pressure of the crowded-back intestines; the surface of the distention is dull at all points; and fluctuation is very distinct at all parts, showing the presence of fluid. All this, taken into consideration with the absence of ascites and the circumscribed appearance of the growth, points emphatically to fluid contained in a cyst; everything indicates an ovarian or broad-ligament cyst, in spite of the free and continual uterine bleeding.

As between the intraligamentary cyst and a free ovarian cyst, we have a tumor which is freely movable for its size, and which does not sink very low into the pelvis, as does an intraligamentary cyst. We therefore decide on the diagnosis of an ordinary unilocular ovarian cyst. Its size precludes, of course, the possibility of saying from which ovary it grows, but we have one hint in this direction,—the patient states that, when the growth was first noticed, it was largest on the right side. It probably grows from the right ovary. One other point in diagnosis before we open this abdomen. The patient comes for relief from two symptoms mainly,—the size and weight, and the bleeding. I am strongly inclined to believe, on account of this bleeding, that we shall find something more than a simple ovarian cyst. Three possibilities enter my mind: first, inflammatory complication; second, small fibroid nodules in the uterine wall; and third, hypertrophic endometritis. If we do not find one or the other of the first two, we shall curette the uterus after removing the tumor. I am inclined to think, from the color of the patient (a negress), it will be fibroid nodules of the uterus, as this condition is very common in the colored race.

A word in conclusion. Is this tumor malignant? I think not. It is too circumscribed and lacks absolutely a nodular condition; its walls are apparently not thick enough, fluctuation is too emphatic, vaginal examination gives no indication of malignancy, and the patient's general health is not such as would indicate a

degenerative disease. She has lost no strength or flesh, and there is no characteristic or suspicious vaginal discharge. For these reasons, I also exclude endometrial cancer as the cause of the bleeding. I think the microscopic examination of the uterine scrapings unnecessary in this case. There are no signs, either physical or clinical, of suppuration, and the blood-count shows no material increase in the white blood-corpuscles.

As I open the abdomen, you see, much to our surprise, universal adhesions between the cyst-wall and the parietal peritoneum. The tumor is evidently going to be difficult to remove, and we shall continue the discussion after the operation, which will require all my attention for the time.

You now see the cyst removed; the abdomen will be closed in the ordinary way and the operation completed. The woman is in prime condition, considering the severe lesions and the amount of traumatism, and I see no reason why she should not make an easy recovery.

We now have before us the tumor, and I wish to call your attention to several points, in view of our previous remarks on the diagnosis. The uterine bleeding is fully accounted for. You will note that the cyst is bleeding at every point. It was absolutely and universally adherent to the abdominal walls, the omentum, the intestines, the uterus, the broad ligaments, and the bladder. The uterus was enlarged to, say, three and one-half inches in depth, a fact which we appreciated and noted in our preliminary examination. The inflammation about and in it, and its enlargement, accounts for the bleeding. The cyst-wall is thick and does not look very healthy, *i.e.,* glistening and opaque like a healthy ovarian cyst; the torn surfaces and the adhesions account for part of this, but not for all. The contents were, as you saw, a dirty septic-looking fluid; septic or not, we were careful not to spill any of it, and therefore do not fear infection from it. The intestines were universally adherent together and to the cyst up to the diaphragm, and had to be separated; in doing which some cheesy nodules were found at points in the mesocolon. You will recall that the patient's history gave absolutely no indication of an attack or attacks of acute peritonitis, or of sepsis at her childbirth, which, from her description, was normal. When all the factors in this case are considered, especial importance being attached to the cheesy nodules

in the mesocolon, I see no reason why the extensive peritonitis should not be considered as of tuberculous origin.

You may ask, "Why did not the examination disclose the adhesions?" The reasons for this are, the lack of a history to indicate inflammatory attacks; the size of the tumor, causing such distention as would in itself render it more or less immovable; and the loose attachment of the thickened and adherent parietal peritoneum to the muscles, giving it more or less mobility together with the attached tumor, both moving together. This is quite frequently the case, and, all things considered, is not surprising. I know of nothing in intra-abdominal diagnosis so difficult oftentimes to determine as the presence or absence of an adhesion. The tumor is unilocular ovarian, and the woman is to all appearances a full-blooded negress. It has been asserted that such a tumor never grows in a full-blooded negress, and even if they do occur, they are quite rare.

A much more rare occurrence, one which I have never before seen or heard of, in fact, is this condition of the cyst. You will observe that near the attachment of the Fallopian tube, which was removed with the cyst and remains attached to it as usual, there is this hole the size of a silver ten-cent piece. The hole perforates both the anterior and posterior walls of the cyst, but does not at any point communicate with the interior of the cyst, although the cavity of the cyst completely encircles the hole. You see that by introducing my finger into the cavity of the cyst through the opening made by the trochar, I am enabled to pass it completely around the hole. Through this hole passed a double knuckle of ileum, the free end of which lay on the bladder in a bunch as large as my fist, and was universally adherent to the cyst and bladder and the anterior face of the uterus; it was adherent also in the canal. In other words, we had complicating our operation, and adding materially to our embarrassment in the removal of the tumor, an incarcerated hernia of the ileum, the ileum becoming incarcerated in the cyst-wall. As I said before, this condition is unique. We shall place the specimen in the hands of the pathologists, and ask them to help us determine both the question of tuberculous inflammation and the method of production of the hernial opening. Two methods of producing the hernia suggest themselves,—pressure-necrosis, and the growth of the cyst about the intestine, and later union of cyst-

walls above it with early obliteration of the points of union. I am rather inclined to the second theory. Pressure-necrosis, from the condition of the whole specimen, seems to me improbable. You will note that at no time was there a history of obstruction, or even of undue constipation. This is quite a common history in generally adherent abdominal viscera, and even occurs in incarcerated hernias, but seems a little strange where both these conditions exist together.

In spite of the extensive adhesions, you note that we used no drainage; a far better plan is to fill the abdomen with salt solution. If your operation has been clean, the blood will take care of itself, provided there is not too great a quantity.

THE TREATMENT OF CASES OF FACE PRESENTATION.

BY ROBERT JARDINE, M.D., F.R.S.E.,

Senior Physician to the Glasgow Maternity Hospital, Examiner in Midwifery to the University of Glasgow, etc.

A FACE presentation is a somewhat rare occurrence; about one case in two hundred labors may be taken as the average. The chin or mentum is the leading part or denominator, just as the occiput is when the vertex presents. As in vertex presentations, we have the four positions, two anterior and two posterior. The best way to look at a face case is to regard it as an ordinary head presentation in which sufficient extension has occurred to bring the chin, instead of the occiput, to be the leading part. If we take a first vertex presentation, L. O. A., and cause extension to take place, we find that the chin naturally points towards the right sacro-iliac joint and the forehead towards the left iliopectineal eminence, the long diameter of the face being in the right oblique diameter of the pelvis. The first, second, third, and fourth face positions are named according as they are converted from first, second, third, and fourth vertex. Thus, L. O. A. gives R. M. P.; R. O. A. gives L. M. P.; R. O. P. gives L. M. A.; and L. O. P. gives R. M. A. The first facial position, or R. M. P., is the most frequent one because the vast majority of vertex cases present in the L. O. A. position.

Face cases may be divided into primary and secondary. In the primary, the head is extended and the chin leading when labor begins. This may be caused by any abnormal condition which will throw the chin up or draw the occiput upward. The chin may be thrown up by a great development of the chest or by a tumor of the thyroid or the thymus gland; and the occiput may be drawn up by a spastic condition of the muscles of the back of the neck. In the secondary, extension has not been present at first, but as soon as the head has commenced to descend the occiput has become hitched on the brim of the pelvis and the chin has been forced

down. A head with a long occiput predisposes to this. It is apt to happen if the uterus is obliquely inclined towards one or the other side, and also in contracted pelves, as in such cases the occiput is very apt to hitch on the brim as soon as descent begins.

Mechanism of Labor in Face Cases.—This is the same as in vertex cases, only, of course, extension and flexion are reversed while internal and external rotation remain the same. As the majority of the cases begin with the chin posterior, long rotation must occur if the chin is to pass forward. If extension is well marked, so as to bring the chin well in advance of the forehead, the leading part is bound to follow the ordinary rule and move forward as soon as it strikes the pelvic floor. On the other hand, if extension is not sufficiently marked to bring the chin in advance of the forehead, the same rule will be followed, and the leading part, or forehead, will pass forward while the chin rotates into the hollow of the sacrum. When this occurs, unless the child is very small, it is almost impossible for the birth to take place by natural efforts. The neck is so short that the bent-back head and the chest of the child must necessarily pass the brim together, and there is not room for this unless the child is very small, or dead and macerated.

Prognosis.—Even if seen early, the risk to the mother is slightly greater than in a vertex presentation, and much greater when impaction has occurred. The risk to the child is considerably greater than in ordinary cases.

Diagnosis.—If the abdominal wall is thin, the diagnosis may be made by external palpation. If the membranes are unruptured, they will usually be found protruding in a sausage shape; the presenting part will be high up, soft, and irregular. In the early stage, before extension has become great, the anterior fontanel and forehead, the orbital ridges, eyes, nose, mouth, and chin can easily be made out. If the membranes are intact, care must be taken not to rupture them until the os is fully dilated, and one must also be careful not to injure the eyes or mouth.

Treatment.—If the chin is presenting anteriorly, the case may be left to nature, provided there is no disproportion between the pelvis and the head; but if the pelvis is slightly contracted or the head is a large one, it should be dealt with either by bringing the occiput down or by version as soon as the os is fully dilated.

If the membrane has ruptured early and the face is moulded into the pelvis, and it is evident that uterine action is not sufficient to finish the labor, forceps should be applied. With the chin to the front, forceps delivery is usually not very difficult. Care must be taken of the perineum, as it is readily torn. In mento-anterior cases the difficulty of delivery is not usually very great, but with mento-posterior presentations we have a much more difficult task. If I see a case early, before or shortly after the membranes have ruptured, and before the uterus is moulded around the child, I put the patient under chloroform, pass my hands in and grasp the occiput, and flex the head so as to convert the case into an ordinary vertex presentation. It may then be left to nature if desired; but, as the patient is under chloroform, she may as well be delivered at once by forceps. In secondary face presentations this can nearly always be accomplished without difficulty, but in primary cases, as a rule, it cannot be done. If I find that flexion is impossible, I always turn at once.

The following cases will illustrate this. The first patient was a multipara, who at her last confinement had been delivered of a still-born child by turning. So far as I could make out, it had been a face presentation. I found the os fully dilated, the membranes intact, and the face at the brim, R. M. P. It was evidently a secondary face case caused by obliquity of the uterus. Under chloroform I had no difficulty in converting it into a vertex presentation and delivering a live child with forceps.

The patient in the second case was a somewhat elderly primipara. The face presented mento-posteriorly. I failed to flex the head, so at once did podalic version. As the head was very large and the pelvis somewhat small, there was considerable difficulty in getting the aftercoming head through and the child was still-born. The extension of the head had been caused by the great development of the chest of the child, which had thrown the chin up. This had prevented flexion of the head.

In the third case a multipara with the face presenting mento-posteriorly had been in the second stage of labor for several hours The face was not jammed into the pelvis and, although the membranes had been ruptured for some time, the uterus was not retracted around the child. I failed to flex the head, probably because it was partially in the pelvis. As there was no retraction, I at

once turned without any difficulty. The arms passed up and one of them lay behind the head. As I found it was impossible to disengage it, I deliberately broke the humerus through and delivered the child alive. The broken bone was put in a splint and bound to the child's body for a week, and it united well.

Schatz's method of converting face presentations into vertex presentations by external manipulations looks well on paper, but it is an exceedingly difficult thing to do. In the mento-posterior cases it is almost impossible to do it, and these are, unfortunately, the ones in which interference is required.

In the cases in which the face is driven down into the pelvis and flexion of the head or version is out of the question, we must wait on nature, for a time at least. We may assist matters, however, by increasing extension and helping the chin to rotate forward. If it is evident that natural efforts are not sufficient, forceps should be applied, and, as a rule, delivery can be effected if the chin has not rotated into the hollow of the sacrum. The most difficult case I ever had to deal with was one in a flat pelvis. The face was jammed down with the chin lying towards the right side of the pelvis, the long diameter of the face being in the transverse diameter of the pelvis. The double-curved forceps would not hold, but I succeeded in delivering with a pair of straight ones. The child gasped a few times, but we could not resuscitate it. It was impossible to rotate the chin forward until we got the head well down on the perineum, as the flattening of the pelvis prevented this. If this case had been seen early enough, the proper treatment would have been Cæsarean section, but it was too late for this when the patient came under my care. When the chin has rotated into the hollow of the sacrum, forceps may be tried, but the chances of getting a live child are very remote unless it happens to be a very small one. In the interest of the mother, craniotomy is indicated, and it should be done early so as to avoid laceration of the parts. The perforator may be passed through the orbit or the roof of the mouth.

While I strongly advocate turning in face cases if the occiput cannot be brought down, it must never be attempted if the uterus is at all retracted around the child. The following case will illustrate this. The patient was a multipara with a normal pelvis and her former labor had been easy. The face presented mento-

posteriorly, but failed to enter the brim, evidently from the large size of the child. Repeated efforts had been made to deliver with various kinds of forceps, but without avail. A ridge across the abdomen below the umbilicus had been noticed, but its dreadful significance had not been recognized. As a final effort, version had been determined upon. A band had been passed into the uterus and a foot grasped, but before any traction could be made the patient collapsed. She was brought to the Glasgow Maternity Hospital from some distance in the country, and died a few minutes after admission, before anything could be done. It was quite evident that the uterus had been ruptured. There was a distinct sulcus running obliquely across the abdomen below the umbilicus. I removed the uterus *post mortem,* and found that the rupture did not extend through the peritoneal layer. After hardening the specimen in formalin, I opened the womb and found that the rupture was of the exact shape of the back of the closed hand. Bandl's ring showed very distinctly as a ledge running around the interior of the uterus; the rupture was below it. Turning would have been a perfectly justifiable method of treatment in the case if it had been done early, but to attempt it after Bandl's ring was apparent could have only one ending,—rupture.

Diseases of the Eye, Ear, and Throat

SYPHILIS OF THE EYE.

LECTURE DELIVERED AT THE WESTERN PENNSYLVANIA MEDICAL COLLEGE.

BY EDWARD STIEREN, M.D.,

Instructor in Diseases of the Eye and Ear, Western Pennsylvania Medical College; Ophthalmologist to the Pittsburg Free Dispensary, etc.

GENTLEMEN,—The several tissues of the eye, its appendages and adjacent structures, may be affected by syphilis. The numerous local lesions that result are usually serious; they nearly always run a tedious course and are prone greatly to impair vision. It may even be the point of initial infection. The eye may be invaded at any stage in the course of this disease.

For convenience and simplicity syphilitic lesions of the eye, with their special treatment, will be considered in the order of the anatomical structures from without inward, independently of the period of the disease at which they occur.

LACHRYMAL APPARATUS.—Stricture of the lachrymonasal duct is due to syphilis in a certain proportion of cases. While pronounced disease of the nasal bones or cartilage may not be present, stricture or displacement of the lachrymonasal duct on one or both sides may be associated with perforation of the septum, or of the floor of the nares, sinking of the nasal bridge, or caries of the turbinated bones,—tertiary manifestations of syphilis. These conditions call for the employment of full doses of potassium iodide and the judicious use of probes. In syphilitic cases the largest probes are most often required; from Number 10 to Number 16 Theobald probes are usually necessary.

Gumma of the skin overlying the lachrymal sac has been reported; the diffuse character of the tumor overspreading the anatomical limits of the sac, the fact that it can be grasped between

the thumb and finger, and the existence of a specific history, establish the diagnosis.

The *lachrymal gland* is singularly free from syphilitic affections, but specific induration and inflammation have been described; in hypertrophy of this gland of obscure etiology, careful antisyphilitic treatment should be instituted before resorting to surgical measures.

Eyelids.—Primary chancre of the eyelid has been observed both in adults and children; the sore presents the same appearance as when situated upon the genitals, and its treatment requires no special remark. If situated upon the cutaneous surface it does not greatly endanger the eye, but if on the mucous surface, or on the caruncula, it becomes more serious. To meet this condition, mercury is to be energetically exhibited and the sore must be kept scrupulously clean; calomel should be dusted on the primary lesion with unremitting frequency until it heals.

Mucous patches, often of considerable size, may develop on both the cutaneous and conjunctival surfaces of the lids. These usually yield readily to weak astringent washes or solutions of silver nitrate, ten to twenty grains to the ounce. Various forms of secondary cutaneous eruptions may appear on the skin of the lids; the eyelashes and eyebrows are often lost simultaneously with the hair of the scalp. Gummata, sometimes attaining the size of a hazel-nut, may appear in the lids and adjacent parts. These lesions belong to the late stages of the disease and require the appropriate constitutional treatment. Drooping of the upper lid (ptosis) is caused by involvement of the third nerve, and will be referred to in connection with paralysis of the extrinsic muscles of the eye.

Conjunctiva.—Syphilis may attack the ocular conjunctiva in the form of sores from primary infection, mucous patches, or gummy growths. All of these lesions are rare. There is a type of ophthalmia affecting anæmic or colloid conjunctivæ called syphilitic conjunctivitis, which appears as a stubborn catarrh, or in the form of granulations similar to those of trachoma. It is not amenable to local treatment, but disappears under the exhibition of protoiodide of mercury or the mixed treatment.

Sclera.—Syphilis invades the sclera under three forms: First, as episcleritis; second, as simple scleritis; third, as gummy scleritis. The first of these is often confounded with a conjunctival

process; the second may appear at any part of the eyeball as a patch of injection, irregular in outline and accompanied by mild subjective symptoms, such as pain, photophobia, or impaired vision. It is inclined to be persistent and rebellious, and usually attacks both eyes. The iris, ciliary body, choroid, vitreous, and cornea may be affected at the same time. The third, or gummy infiltration, is easily recognized by signs of interstitial deposit and redness; it tends to spread to deeper parts, and may ultimately lead to destruction of the eye. In addition to the energetic administration of bichloride of mercury or inunctions, the local employment of atropine, hot compresses, leeches to the temple, and protection of the eyes by dark glasses or residence in a darkened room should be rigidly adhered to.

Cornea.—Interstitial keratitis is a chronic inflammation of the entire cornea, resulting in universal, thick haziness, with or without elevation of the surface. In most instances it is due to inherited syphilis, evidence of which is present in about seventy per cent. of the cases, which occur most frequently between the ages of five and fifteen years. After a few days of slight ciliary congestion and lachrymation a faint cloudiness appears, usually near the centre of the cornea. In two or three weeks this spreads until the whole cornea is invaded by a diffuse haziness, veiling or completely hiding the iris. The subjective symptoms of irritability and photophobia are more pronounced in strumous children who are at the same time syphilitic. Ulceration occurs occasionally; iritis and formation of posterior synechiæ are not infrequent concomitants. In the course of time, the length of which depends upon the treatment, the cornea begins to clear, usually from the periphery. Perfect restoration of transparency is rare, although the remaining haze may be slight. The subjects of typical forms of this phase of the disease are familiar pictures in all eye clinics; the dwarfed stature, the coarse, flabby skin, the sunken nasal bridge, the scars at the angle of the mouth, and the Hutchinson teeth, indelibly stamp the heritage of the patient.

Prognosis.—From six to eighteen months are usually required for the development of the various stages of the disease. The second eye is almost invariably attacked in from a few weeks to two months. Relapses are frequent, not only of the corneal disease but also of the accompanying lesions of the iris and retina.

Treatment.—In the first stages *all irritating applications are harmful.* Atropine should be systematically employed to maintain mydriasis, prevent iritis, and allay inflammation. The severity of the inflammation demands the continuous use of hot fomentations; tenderness of the ciliary region may be relieved by a leech applied to the temple. The eyes must be protected from the light either by dark glasses or by residence in a darkened room. The best general medication is a long-continued course of mercury. In children certainly, and probably in adults also, the most satisfactory method of administering this drug is by inunction, one drachm of mercurial ointment being rubbed into the skin once or twice a day. During the period in which the inunctions are being employed cod-liver oil may be given; later, bichloride of mercury is a valuable remedy, and it may be advantageously combined with the tincture of the chloride of iron, as many of the patients are anæmic. When all irritation has subsided, clearing of the opacity may be hastened by the use of a salve of the yellow oxide of mercury, three grains to two drachms, in conjunction with massage of the cornea; or, what I have found of distinct advantage, cataphoresis of the above-mentioned ointment with the galvanic current. Calomel or powdered aristol dusted into the eye has also been found useful.

The Iris.—There are two affections of the iris which result from syphilis—paralysis of the sphincter of the pupil, causing mydriasis, and inflammation.

Mydriasis occurs under two conditions. In the one it is associated with evident paralysis of the entire third nerve or some of its branches; so that, besides dilatation of the pupil, there may be ptosis, divergent strabismus, and diplopia. Another condition in which mydriasis appears does not present any sign of lesion of the third nerve, so far as other twigs are concerned, but appears to be associated with obscure changes in the brain, or at the base of the skull, which may not at the time declare themselves by noticeable symptoms.

The *treatment* of syphilitic mydriasis calls for the local use of a solution of the sulphate of eserine or of the muriate of pilocarpine, and the internal administration of large and increasing doses of potassium iodide and strychnine. Faradism to the closed lids has also been recommended.

Inflammation of the iris, or iritis, is a disease uncommon during

the first fifteen years of life and during old age, being most frequently met between the twentieth and the fortieth year, and affecting relatively more men than women. Syphilis is without doubt the most common predisposing cause of iritis, and if hereditary syphilis be taken into consideration the proportion it accounts for is about sixty per cent.

The *symptoms* of an attack of iritis may be considered subjectively and objectively. Objectively, we recognize a change in the color of the iris, due to congestion and effusion of lymph and blood corpuscles into its substance, a blue or gray iris becoming greenish and a brown one presenting a muddy appearance; pericorneal injection, a fine pink zone surrounding the cornea, due to the engorgement of the episcleral vessels; myosis, or contraction of the pupil, due partly to hyperæmia of the iris and partly to irritation of peripheral nerve filaments. The reaction of the pupil to light is diminished or lost. Posterior synechiæ may be present. In nearly all syphilitic cases there is a formation of gummatous nodules on the surface of the iris. There may be haziness of the cornea and the aqueous humor is often more or less turbid, or may contain pus, blood, or exudate.

Minute opacities are often found on the inner surface of the cornea, especially on its lower half; they are in part precipitations, but also result from participation of the membrane of Descemet in the inflammatory process. The ciliary body and choroid are still more frequently affected; all obstinate and persistent cases of iritis pass into iridochoroiditis. Especially is this true when the plastic exudation is copious or when the pupil has not been dilated. At times the vitreous becomes hazy, the retina suffers, and often the eyeball becomes soft to the touch, or even reduced in size. Vision in these complicated cases is seriously impaired. The formation of a tough fibrous membrane across the pupil and thickening of the anterior capsule of the lens are consequences to be looked for in badly treated or severe cases; as a result of iridochoroiditis, cataract not seldom arises. The subjective symptoms generally present are pain, disturbance of vision, tenderness of the globe, photophobia, and lachrymation.

The *treatment* in uncomplicated cases consists in the local use of atropine and warm fomentations, protection of the eyes from light, and the internal administration of small divided doses of

calomel. The pain is best met by the exhibition of full doses of salicylates. Local bloodletting from the temple may be necessary.

Vitreous Humor.—Inflammation of the iris and ciliary body and choroid is often followed by the appearance of opacities in the *corpus vitreum*. These are due either to effusions from the surrounding vascular tissues or to proliferation and degeneration of the cells of the vitreous. The anterior and posterior parts of the vitreous are most frequently affected, the centre rarely. The opacities present every variety of form,—molecules, fibres, tangled nets, flakes, and membranes,—and usually develop slowly.

In the *treatment* of vitreous opacities, a favorite preparation, used in combination with calomel and potassium iodide, is Zittmann's decoction, in wineglass doses thrice daily or oftener. The efficiency of this agent may be augmented, if the condition of the patient justifies their employment, by full doses of pilocarpine or jaborandi, marked improvement following the perspiration induced by these drugs.

Choroid.—The similarity in structure of the iris and choroid predisposes to a corresponding similarity in their morbid processes, but in many instances the refractive media become so soon deeply clouded that it is impossible to observe the changes which occur in the choroid. A form of choroiditis which may take place without involvement of the iris or visible hyperæmia of the globe is known as *acute choroiditis disseminata*. Illuminated by the ophthalmoscope, the vitreous is seen to be faintly hazy, but through it may be discerned upon the posterior wall of the eye a number of small isolated specks of a light yellow color. These specks occur most frequently near the equator, but may also be situated at the central part of the fundus. They are unmistakably elevated, and in some instances a retinal vessel may be seen to pass over them. The optic nerve is always hyperæmic, but is not infiltrated. This picture, after two or three weeks, begins to undergo alterations. The yellow specks grow fainter, and pigment is deposited around their borders. After a time, with the more complete disappearance of the primary deposits, the subjacent choroid is found to have become thin through destruction of the epithelium; the stroma of the choroid also is ultimately absorbed, leaving only a dead-white patch (the sclera bared of choroid and retina), with a border deeply fringed by black pigment. In severe cases and those of long standing the appearance

of the eye-ground is most striking: circular and oval white spots with black edges are clustered thickly over its surface, presenting a brilliant contrast to the red color of the choroid. There may be also patches of lighter red, indicative of thinning of the membrane. Vision is always much impaired, and may be entirely lost. These lesions may be most extensive at the periphery of the choroid; on the other hand, the central region of the fundus may alone be affected, and this virtually means blindness.

Another and slower syphilitic process affecting the peripheral parts of the fundus is atrophy without the previous deposit of lymph. The wasting of the choroid is gradual, and the patches present a light red surface bordered by white, the whole bounded by a ring of dark pigment; where the patch is light red a portion of the membrane still remains. These patches develop very slowly and insidiously, and they are sometimes discovered in an eye supposed to be perfectly sound. Direct vision may be normal, but the visual field will be encroached upon.

Treatment.—In the cases first described, during the stage of exudation, a somewhat active treatment would be proper, as the application of two or three leeches or the artificial leech to the temple and the seclusion of the patient for twenty-four hours afterwards in a darkened room. This may be repeated in five or ten days. Dark glasses should be worn. The constitutional treatment of syphilis should be pushed with as much energy as the tone of the system will bear. In the chronic form of choroiditis only the milder methods of constitutional treatment are appropriate.

Retina.—In syphilitic retinitis there is slight haziness and œdema of the retina, indistinctness in the outline of the vessels and of the optic disk, and hyperæmia both of the retinal vessels and of the optic nerve. The deeper part of the vitreous is hazy. The peripheral part of the retina may remain free from perceptible change, the disease being usually confined to the central region, including the nerve; sometimes, however, it is more narrowly localized in the vicinity of the yellow spot itself. This form of inflammation may attack one or both eyes, and may pass from one eye to the other. It may run a rapid course, say for three or four weeks, or it may persist for several months. It does not usually, but it may cause permanent damage to sight. The subjective symptoms consist of occasional flashes of light at the beginning of the disease,

and subsequent dimness of vision; there is no pain or lachrymation, but little photophobia, and no external hyperæmia.

Treatment need never be energetic: protection against bright light by dark glasses, abstinence from use of eyes, and the application of the artificial leech according to the usual rules,—two to four times, at intervals of a week,—constitute the most satisfactory local treatment. Reliance must rest mainly upon the constitutional treatment.

Other forms of retinitis are associated closely with the choroidal changes, already described. There is another syphilitic manifestation, however, which appears to be due to localization of the disease in the retina. The ophthalmoscope reveals a gray opacity surrounding the nerve entrance and extending in lines along the vessels; the papilla is discolored and cloudy, and appears as a yellowish-red, oval body seen through a foggy medium. The veins are darker and fuller than normal; the arteries are not, as a rule, materially changed. Exudations may follow the course of the vessels, and extravasations of blood, usually round in shape and due to endarteritis, may occur. Thrombi may be formed, but on the whole, hemorrhages in syphilitic retinitis are rare. Treatment is the same as in the other form.

Optic Nerve.—*Neuritis optica* may occur as a local manifestation of the general disease, or it may be caused by pressure due to the formation of an intracranial gumma. Hence, two varieties are recognized: first, that which primarily affects the outer orbital extremity of the nerve, and second, that which is set up by intracranial causes. In both varieties the retina may be implicated to a greater or less extent. The distinction between the two classes of cases cannot be made with certainty by the ophthalmoscope alone, but the question of the intracranial or extracranial origin of the lesion always presses for solution.

The *symptoms* which appear vary according to the degree of the inflammation and according to its stage: First, in simple cases nothing is seen but redness of the nerve surface and a little fulness of the central vessels, with scarcely any blur of the edge of the disc or of the tissue. Second, in other cases the nerve is swollen to an extreme degree, infiltrated and opaque, often striated, its color is red or gray, its border wholly or partially obliterated, its vessels tortuous and turgid. This is the so-called "choked disk," and its

cause is usually intracranial. Third, in certain other cases of brain syphilis the optic nerves become affected and exhibit on inspection only a little white color and a "woolly" appearance, with slight blurring of the edge, the vessels being small. There may be a doubt, perhaps, whether a faint degree of hyperæmia may not have preceded this condition; but if it has, its duration has been extremely brief. In these cases, as well as in the ones previously mentioned, it is of the utmost importance to determine the extent of the field of vision. It will be found almost invariably to be curtailed at some part; irregular hemiopia is often present or a quadrant of the field may be wanting.

Prognosis in these affections is never good, but a valuable degree of vision is often preserved or recovered.

Treatment consists in the exhibition of increasing doses of potassium iodide, strychnine until the point of tolerance is reached, phosphorus in some form, and mercury and iron when indicated.

Affections of the Motor Nerves.—An extremely common effect of syphilis is disturbance of the function of the motor nerves of the eye; one muscle, or any number of the muscles, may be paralyzed. Inasmuch as the third nerve supplies four of the muscles which move the globe, the eye, when this nerve is attacked, is most helpless; but separate twigs may be singled out while others are undisturbed. If all the branches of the nerve are affected, the eye stands at the outer palpebral angle, is incapable of motion up or down, and cannot turn inward farther than the median line; the upper lid droops and cannot be lifted. The eye can be carried farther outward by the external rectus than under normal conditions, and by the action of the superior oblique it can still make some rotary movements. The pupil will be in medium dilatation and the function of accommodation paralyzed. Diplopia will not commonly be noticed, even if the lids be opened, because the two images are so far asunder as not to attract attention. As the nerve begins to recover and the eye to regain mobility, diplopia will become annoying and the images will be crossed. If the sixth nerve is paralyzed, *i.e.,* paralysis of the external rectus muscle, the eye stands in abnormal convergence, because the abductive power of the external rectus is destroyed. Double images are then correspondent (homonymous)and are most annoying for distant objects, while an object brought near the eye may be seen correctly.

It was my privilege last term to be able to present a very interesting and unusual case. The patient, a man about thirty-five years old, who gave a rather meagre syphilitic history, became suddenly blind for near objects, and in order to see distant objects distinctly it was necessary for him to turn his head far to the left. Examination revealed paralysis of the left external rectus, with no laming of any of the other extra-ocular muscles of either eye. There was in addition, however, complete *ophthalmoplegia interna,* paralysis of the ciliary muscle,—the muscle of accommodation,—in both eyes. After a prolonged course of potassium iodide combined with strychnine, the faradic current over the affected external rectus, and the instillation of eserine, he regained full control of the motion of the affected eye and of accommodation, so that he was able to read the finest print without the aid of glasses.

If the fourth nerve is paralyzed, superficial inspection may fail to recognize the defect in mobility, but it may be detected with certainty by careful study of the double images. To do this, a lighted candle is chosen for an object and a piece of red glass placed before one eye. There may be no diplopia in the field above the horizontal line, but, as the eyes descend, double vision occurs, one image (the false one) being below the other; as the object is carried to the temporal side of the affected eye, the images, besides being one above the other, separate laterally, the false one being farther to the nasal side. Patients who, from any kind of paralysis, have diplopia, are much disturbed thereby, sometimes having nausea and headache, while, to use their eyes, they must either shut one or correct the double vision by some twist of the head, or by means of properly adjusted prisms.

During the early stages of paralysis of the extrinsic muscles of the eye, the proper treatment is counterirritation to the temples, the faradic current, and constitutional remedies. If after the lapse of a number of months motion is still imperfect, the use of prisms or the performance of tenotomy or advancement of the muscles must be resorted to. The treatment of internal ophthalmoplegia has been given in the case cited.

Syphilitic periosteal inflammation of the orbit does not often occur, but some symptoms which it causes are worthy of attention. If it affect the deep parts of the cavity, it may cause disturbance in the function of some of the muscles, and hence diplopia; or, if

attended by sufficiently profuse effusion, it may produce exophthalmos.

As to gummy tumors in the orbit, nothing special need be said; that they may displace the eyeball, and otherwise interfere with its functions, is self-evident. There may be nodes upon the walls of the orbit, and necrosis or caries is not infrequent; abscesses may form and a fistula often remains; frequently the lids become distorted by adhesions of the skin. Sometimes these inflammations are attended by severe constitutional disturbance, and there is also danger of involvement of the adjacent cerebral structures.

Treatment must be vigorously antisyphilitic. Surgical intervention is often required to remove dead bone, to give vent to pus, to correct ectropion, etc.

FOLLICULOSIS; TRACHOMA; REMOVAL OF CATARACT.

CLINICAL LECTURE DELIVERED AT THE LOUISVILLE HOSPITAL COLLEGE OF MEDICINE.

BY P. RICHARD TAYLOR, M.D.,

Dean of the Faculty and Professor of Materia Medica, Therapeutics, Otology and Laryngology, and Clinical Professor of Ophthalmology, in the Hospital College of Medicine, etc., Louisville, Kentucky.

GENTLEMEN,—I shall show you to-day five cases of conjunctival disease, in all of which the casual observer would make the diagnosis of trachoma; but, upon closer observation, I will make a diagnosis of folliculosis in the three younger patients—girls aged nine, twelve, and seventeen—and of trachoma (granular lid or conjunctivitis) in the young man of twenty-four and the old lady of sixty-three.

I first call attention to the three cases of folliculosis. You notice considerable puffiness of the lids, especially the lower lids, in the two older girls; also that the ocular conjunctiva is but little, if at all, involved. I find the cornea normal. (Fig. 1.)

Everting the upper and lower lids, we find numerous pale-red bodies in the conjunctiva, larger in the folds and diminishing in size as they invade the tarsal area. These small bodies are beneath the epithelial layer of the conjunctiva; they are enlarged lymphatic follicles, composed of lymphatic cells, a little connective tissue, and a very limited blood supply, which accounts for the slight annoyance from this condition until it has advanced so far that the secretions have become excessive and tend to agglutinate the lids at night.

Folliculosis is a contagious disease, found most frequently in delicate, anæmic, and lymphatic children, and especially among those living in crowded tenements provided with imperfect sanitary appliances, irrespective of their physical condition. These children are close friends, and, I have no doubt, have transmitted the disease from one to the other, the youngest taking it last; in fact, she did

not complain of any inconvenience, and did not come to the clinic for the purpose of being treated.

Having rendered the conjunctiva comparatively insensible to pain by the instillation of two drops of a four per cent. solution of cocaine, I evert the upper lid, pouting it outward and downward over the lower by passing one blade of the ciliary forceps beneath and the other above the everted lid. Marked compression is produced, and the closed forceps is made to strip the conjunctiva, forcing from the follicles all accumulations, as the firm pressure breaks up the little neoplastic bodies. Both the upper and the lower lids should be rolled if involved; and this procedure should be repeated at least once every week. I will supplement this treatment by prescribing a salve composed of yellow oxide of mercury, four grains, and vaseline, one-half ounce, to be put into the eyes night and morning; and shall ask these patients to return to the clinic one week from to-day.

I will next direct your attention to the cases of trachoma. Upon everting the lids you may observe that the palpebral conjunctiva is remarkably roughened, nodular, and very much swollen; that the cornea of each eye is involved; that there is extreme pannus of the left cornea, with an extensive ulcer at its apex, which, in this acute stage, has reduced the young man's vision to mere perception of light; that there is a large ulcer of the left cornea a little to the nasal side, which has progressed until there is danger of perforation. Photophobia is excessive, due to the corneal ulceration; and there is iritis in the right eye, due to extension of the irritation to the iris.

Though the disease here is of comparatively recent origin (only about fifteen months' duration), we find all the complications usually met with in such cases. Routine treatment will not meet the indications. Usually silver nitrate, varying in strength from a two per cent. solution to the mitigated stick, is applied. Copper sulphate and yellow oxide of mercury are used with benefit; but I shall direct my treatment to relief of the pannus and to the corneal ulcers, which endanger his vision. I call your attention especially to the swelling of the lids and orbital tissue, and point out further that the palpebral fissure is not shortened, a condition that occurs when trachoma is of long standing.

To relieve the corneal complications, I must remove the friction and pressure caused by the upper lids of both eyes. This I do by

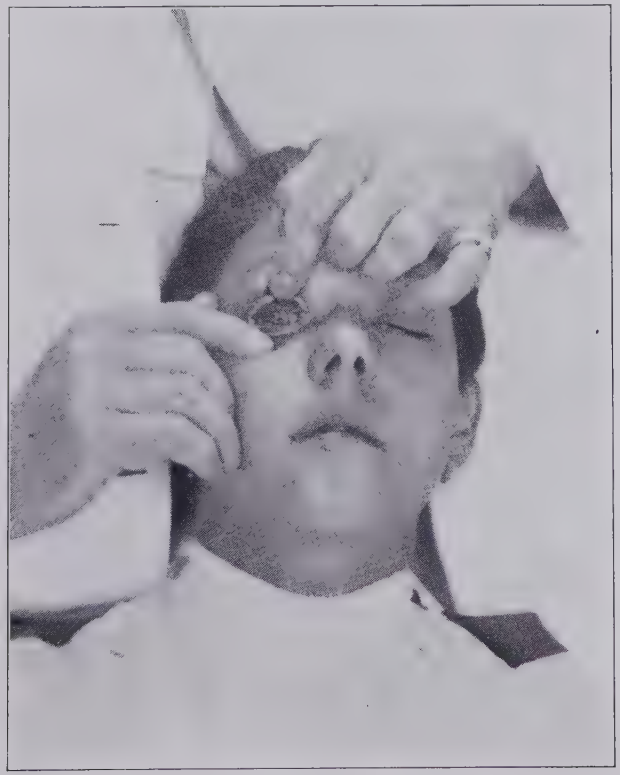

FIG. 1.—Eyelids everted, showing folliculosis.

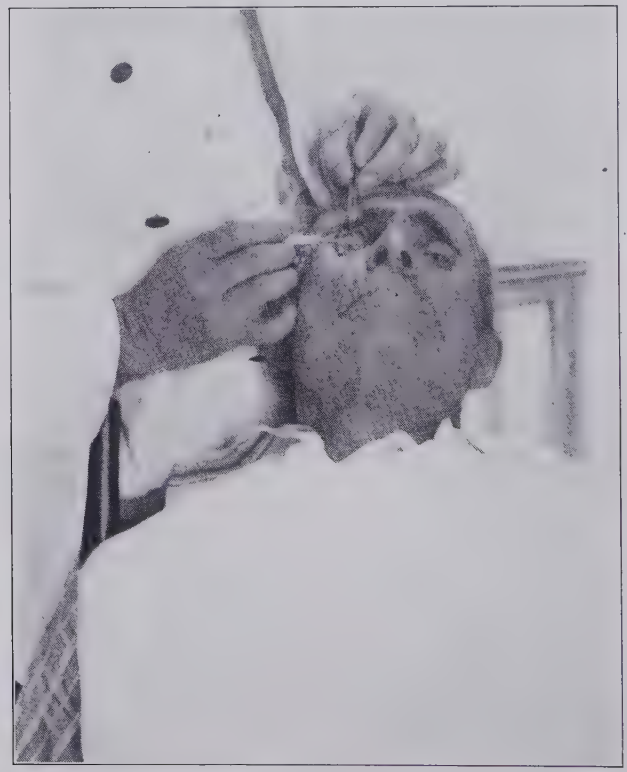

FIG. 2.—Operation for cataract complicated by trachoma.

everting the upper lid, placing a little roll of gauze, one and one-half inches long and about the size of a lead-pencil, back of the upper lid so that it will tend to remain everted. I thrust this needle armed with coarse braided thread through the lid and pass it on through the tissue of the brow. I make two stitches in the upper lid of each eye, tying them with but little tension, so that there may be less tendency to cut through. The roll of gauze will be held in position behind the everted lid, and should remain until the stitches are removed.

Having stitched the lids to the brows, I now apply with the utmost care a solution of nitrate of silver, thirty grains to the ounce, to the everted lids, using gauze so as to let none of the solution reach the cornea. This application should be made each day.

An ointment of yellow oxide of mercury, eight grains to the half-ounce of vaseline, should be freely applied to the lid and cornea, with frequent hot fomentations over the brow and temple. The lids should be kept stitched, and this treatment continued until the ulcers have healed, by which time great improvement will have taken place in the lids. After the lids are released, the usual treatment for trachoma can be employed. I will in this case allow them to remain everted at least one week, if not longer.

This old lady is suffering from chronic trachoma of long standing. The palpebral conjunctiva is a little roughened and very much thickened. The palpebral fissure in each eye is markedly shortened. She did not apply for treatment of the trachoma, but for relief from the rapid loss of vision. I find she has a ripe cataract in the right eye and one well advanced in the left. The vision of the right eye has been reduced to mere perception of light. Relief can be had only by removal of the cataract, to which she has consented. A drop of a one-half of one per cent. solution of atropine sulphate has been dropped into each eye. The pupil is completely dilated. After anæsthetizing the eye with a few drops of a four per cent. solution of cocaine, I will introduce the eye-speculum in the usual way and begin the operation. (Fig. 2.)

I have, you will notice, grasped the conjunctiva with the mouse-tooth fixation forceps just at the sclerocorneal juncture, above, instead of below, as is usually done. This makes it easier to prevent the mishap of cutting away a portion of the iris in cases in which the orbit is deep and the eye receding. If, when the knife is passed through the anterior chamber, the aqueous fluid should escape and

the iris float in front of the knife, the sclerocorneal juncture may be raised by making traction with the fixation forceps; then the iris will drop below the edge of the knife. This complication could not be avoided so readily if the fixation forceps were placed at the lower edge of the cornea instead of the upper.

I now pass a von Graefe knife through the sclerocorneal juncture, cutting the flap of the cornea above, and avoiding the iris, so as to have a normal pupil; the incision being completed, I introduce a cystitome and tear extensively the anterior capsule of the lens. The more tearing of the capsule at this time, the less likely the necessity for subsequent needling.

The lens is now ready to be extracted. I hold the fixation forceps firmly against the globe and make gentle pressure against the lower sclerocorneal juncture, which lifts the upper edge of the crystalline lens into the dilated pupil. Continuous pressure forces it out through the corneal incision. After the lens has been removed, a one per cent. solution of eserine is dropped into the eye; a few drops will quickly contract the pupil. Care should be exercised, in removing the speculum, not to make pressure on the globe, so as to avoid causing escape of the vitreous fluid.

After the pupil has contracted, the eye should be carefully closed, and a piece of absorbent cotton, just large enough to fill the orbit, should be placed over the eye and held in position with a strip of adhesive plaster. No pressure or bandage is needed; the upper lid acts as a splint to the wound, and if the eye is kept closed the edges of the wound will remain in apposition.

It has been my custom to look at the eye in from three to five hours after the operation, and each day thereafter until the dressing is permanently removed, which is about the fifth or seventh day. I shall pay particular attention to this case, owing to the fact that it is complicated with trachoma. There are two points in this operation to which I wish especially to call your attention: first, the position of the fixation forceps; second, the removal of the dressing and the close inspection of the eye from three to five hours after the operation and each day afterwards until the dressing is discontinued. I have often replaced a prolapsed iris during the first twenty-four hours after the operation, which could not have been done if the dressing had been left intact for from five to seven days.

ACUTE MASTOIDITIS; ITS DANGERS, ITS IRREGULARITY IN SYMPTOMS, AND THE QUESTION OF TREPHINING.

BY ARTHUR AMES BLISS, A.M., M.D.,
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Such close anatomical relationship exists between the cavity of the middle ear, the mastoid antrum, and the middle and posterior cranial fossæ that it is surprising that inflammation within the middle ear does not, as a very common occurrence, produce inflammation of varying degrees of intensity within the cavity of the cranium. Not only are the little chambers of the middle ear and mastoid antrum very close to the brain, but the roads by which inflammation can advance from them to the neighboring subdural or even cerebral tissues are numerous and direct. At best the tegmen tympani and the roof of the mastoid antrum are separated from the middle cranial cavity, beneath the sphenotemporal lobes, by an extremely thin layer of bone, reduced in many individuals almost to tissue-paper thinness, and many instances have been noted, suggesting false economy in nature, in which the roof of the middle-ear cavity is not closed by any overlying shell of bone, its mucous membrane lining being in direct contact with the dura mater. The petrosquamous suture sometimes never closes, and a permanent fissure is left between the middle ear and the middle cranial cavity. By means of the fenestra ovalis and the fenestra rotunda access is direct to the vestibule and cochlea, and thence by the internal auditory canal to the posterior cranial fossa. Indeed, the small foramina by which pus can find its way from the middle ear and the labyrinth to the posterior fossa are so numerous that one wonders why cerebellar abscesses do not occur more frequently. The vascular system also offers a convenient road for the entrance of infecting material from the ear to the brain. The mastoid vein enters the lateral sinus. Inflammation within the mastoid cells may cause infection to reach the sinus by this tract, there to

form phlebitis of this sinus, with resulting thrombosis, local abscess, and general blood-infection; or, the suppurative process within the cells may involve the wall of the lateral sinus by direct necrosis, and thus produce the same results.

The middle ear so often becomes inflamed, filled with an exudate, and suppurates, the attack terminating happily when the pus is discharged into the external auditory canal by means of artificial or natural puncture of the drum-head, that we forget the instances where a different course has resulted. The signs of danger which attend the more serious or fatal cases are too often not noted, or are not given the attention they deserve. This is especially apt to be the case in those middle-ear suppurations involving the mastoid which occur during severe fevers, as, for example, typhoid, scarlatina, measles, or even cases of grippe.

G. S., a boy fourteen years of age, has suppurative otitis media after a severe "cold in the head" (grippe?). During the week following the onset of the otitis, the temperature reaches 104° F., with rapid falls and rises; the ear has been streaming with pus during the whole week. On pressure, there is considerable pain over the mastoid process, with redness and a trace of œdema. The mastoid cells are trephined to the antrum, and the subsequent history is that of the wound only, for the temperature falls at once to normal.

Another boy, T. L., nine years old, has an attack of otitis media following grippe. From the auditory meatus of the affected ear a very small quantity of a seromucous discharge exudes, entirely different in character and amount from the otorrhœa noted in the case reported above. The temperature is 100° F.; the mind is dull and heavy. There has been no earache, except at the onset of the attack. Pain can be elicited, on pressure, over the region of the mastoid antrum. There are œdema and redness over the same area. The symptoms are persistent. Trephining the cells results in a "dry" operation, as no pus is found, although the cells are opened to the antrum and explored in all directions. At the first dressing, two days later, pus is flowing freely from the region of the antrum. The symptoms are all favorable and recovery is rapid.

A boy, A. K., seven years old, contracts a cold while skating. Severe earache develops. One week later the aural pain is still a feature of the case, but the drum-head does not indicate the presence of a large amount of exudate in the middle ear. The mastoid is

tender but not swollen. The child is irritable, emotional, varying from hysterical excitement to sleepy dulness. The temperature is variable, with high upward springs to 104° F. and quick falls almost to normal. Puncture of the drum-head permits the escape of a few drops of bloody serum. The cells are trephined to the antrum, a mere trace of pus being found. At subsequent dressings a slight amount of a seropurulent discharge is noticed in the antral region. The bad symptoms are not relieved. Coma develops; the case is hopeless. We trephine over the sphenotemporal lobes, and expose the tegmen from within the cranial cavity, finding evidence of leptomeningitis, but no pus. Death occurs. What would an autopsy have revealed? Possibly nothing more than has been learned already. The whole train of symptoms has developed from the attack of otitis media.

During convalescence from typhoid fever, H. W., a colored man, twenty-three years of age, develops otitis media. Early rupture of the drum-head is followed by a profuse otorrhœa, lasting many weeks. Mastoid pain, swelling, and œdema did not appear until one month after the patient had completely recovered from the typhoid fever. It was then necessary to trephine, a greatly disorganized mastoid being found at the operation.

A man forty years of age is brought to the German Hospital comatose and dying. No details of his history can be learned. It is said that an operation for mastoiditis was performed in another city about three weeks ago. The after-treatment appears to have been *nil,* as the wound of this operation, a mastoid trephining of the left side, is in a very filthy condition. We curette out the flabby granulation-tissue, and clean the tract, finding the walls of the cells softened and broken down, especially at the tegmen. At the autopsy on the day following, we find the trace of a large subdural abscess occupying most of the left cranial fossa. The roughened condition of the cranial floor indicates that the lesion is not of immediate origin. A sinus leads directly to the mastoid antrum, while a second sinus has opened upward from the cochlea. At the posterior border of the pons, on the left side, involving the neighboring part of the medulla, is a large cerebellar abscess which has ruptured, and the whole posterior cranial fossa is flooded with pus. The abscess appears to have formed by the passage of pus from the internal auditory canal.

These few histories of cases may stand as types of those seen by every one who has much to do with aural surgery. They are all taken from acute cases of mastoiditis, except the autopsy, which I introduced as a picture of wreck and disaster that illustrates the ravages of this affection when it has advanced to its limits of evil. The reader will note, without my comment, the varied histories of these cases. There appears to be no fixed rule in the courses pursued; nor, apparently, could it have been predicted that one would be a simple case yielding to rational surgical measures, while another advanced progressively to a fatal end in spite of the observance of all indicated treatment. It is extremely suggestive to note, as illustrated by one of the cases here reported, how frequently it happens that, although pus is not found at the moment of trephining, even in the deepest cells, it does flow freely within a day or two days later. May not this discharge come from an already forming subdural abscess? A sinus need not be large to admit much pus to the cranial cavities; in the autopsy reported, the sinus from the mastoid antrum was not to be seen till sought for by the probe. In opening the cells even to the extreme depth, we could not always find such a sinus, but the fact that copious drainage, with subsidence of all symptoms, occurs on the day following an apparently "dry" operation, would lead one to believe that such subdural collections may be not uncommon, and may be thus drained.

Acute mastoiditis is in itself a dangerous condition; and owing to the irregularity of symptoms in different cases, the question when to resort to trephining, or when it is safe to refrain from opening the cells, is often veiled in uncertainty. Patients do recover in many instances without an opening of the cells; yet serious chances are taken with life in delaying the opening, when indications point to its requirement. No small responsibility rests with the surgeon, who must balance the pros and cons and decide this question, for a case does not always terminate with trephining; in spite of prompt action in this regard, the cranial cavity may already have become involved. Especially is this true in those puzzling cases where the phenomena of the attack lack the usual frank course of symptoms, and where we are dealing from the beginning with what might be called a "dry case," marked by the absence of free discharge from the tympanum, by great variations in tem-

perature, by very little swelling or other local signs over the mastoid, and by bizarre mental states—excitement, dulness, hallucinations—which are of brief duration in the beginning, but which cannot be ignored.

Certain indications, however, continuing for several days, may be relied upon as of value in guiding the surgeon to favor an opening of the cells: first, steady, persistent pain over the mastoid process at the region of the antrum, this pain being of even greater import if it extends along the line of the lateral sinus and base of the skull; second, the existence of œdema; third, the patient's temperature, great importance being given to a steeple-like chart with sudden rises and falls. These symptoms may or may not be accompanied by certain other manifestations, whose existence may serve to determine the urgency for operative measures. Such manifestations are vertigo, nausea, and vomiting, double vision, and mental excitement of an emotional character, or a state of dulness and apathy. These conditions will hasten our determination to act, and we need not await their development before acting. The three indications I mentioned first may be watched for a few days, but should not be allowed to persist for a longer period without regard to their warning.

The trephining of the cells should, in all cases, aim to open the mastoid antrum and give it drainage. It is not necessary to do a complete Stacke-Schwartze operation on cases of recent origin, but the cells should be explored, so that no doubt is left that some one cell or group of cells, isolated from the others, may be inflamed or in process of suppuration, especially in the petrous or petrosquamous group of cells.

Although mastoiditis will, in some instances, develop during the most carefully conducted treatment of an otitis media, its prevention is undoubtedly very often due to proper care of the affected middle ear. I would urge the importance of regarding otitis media as something more serious than a merely annoying phenomenon, like toothache. It may, in every instance, be the onset of a train of conditions that will end in the patient's death; yet, the early treatment is well within the physician's skill, without the aid of the specialist. In the onset of an acute otitis media very warm boric acid solution (from ten to fifteen grains in one fluidounce of hot water) douched, not syringed, into the external auditory canal

of the painful ear, followed by the application of dry heat, is the safest and most effective means of relieving the earache. Opiates should be avoided, as they mask symptoms whose development we should watch. The appearance of the drum-head will indicate if need exists for paracentesis; fortunately, nature does this operation early. In most instances, therefore, we see the wisdom of avoiding the introduction of oils and fermentative substances into the auditory canal, which can readily infect the middle-ear cavity through the opening thus made in the drum-head. Catarrhal states of the nasal and nasopharyngeal mucous membrane require a cleansing but non-irritating treatment, whose aim is to overcome the turgescence which is causing nasal occlusion, and to render the tract free from sepsis. All operative work on the nose and nasopharynx is out of place during the acute stages of an otitis media. During the early days of an otorrhœa, while the discharge is still clean, free from pus, and almost entirely serous, the external auditory canal should be dried gently with absolutely clean absorbent cotton, and the meatus closed by a pledget of clean cotton, or by one made from a narrow strip of boric acid gauze, placed loosely in the concha of the auricle, and renewed sufficiently often to have the dressing clean and dry. This is not the time for syringing with hydrogen dioxide or other antiseptic washes, or for insufflation with powdered boric acid. All that may come later, to be used then with due care, if the attack does not terminate in this early stage and the inflammation advances to a suppurative otorrhœa. Inflation of the middle-ear cavity by Politzer's or Valsalva's methods, or by catheterization, must not be practised in the acute stages of an otitis media, and the patient must be cautioned against violently blowing the nose so that the ears feel the pressure of air thus forced into the Eustachian tubes. When the inflammation begins to extend from the middle ear and mastoid antrum to the cells, we are told in most of the books—indeed, it seems to be the stock-in-trade information —that we are to place an ice bag or other cold dressing over the mastoid process. We cannot ignore the wisdom of our teachers, of course; but I myself have had such unsatisfactory experiences in so many cases where I have faithfully used the dry cold applications, that I have discarded them, and now use dry heat instead.

A practical point in the study of a mastoid involvement is the appearance of the external auditory canal. When the cells are

inflamed to the extent of suppuration, it will commonly be noted that the upper and posterior walls of the auditory canal are swollen immediately in front of the tympanic ring. Care must be taken to distinguish a furunculosis of the external auditory canal from an inflammation of the mastoid cells; in furunculosis we not infrequently have swelling and œdema along the line of attachment of the auricle with the head, sometimes extending over the mastoid and squamosa for a considerable distance. As furunculosis very often attends a suppurative discharge from the middle ear, its development sometimes raises a question of uncertainty as to the real cause of the mastoid swelling and œdema. As a rule, the mastoid process will bear firm palpation, percussion, and pressure in furunculosis, although any movement of the auricle itself is attended with pain. Such cases are puzzling, and the error of supposing them to be of true mastoid origin is a very practical matter that must be considered. The rather discouraging observation of Hippocrates, that life is too short for completeness in our art, that "experience is fallacious and judgment difficult," has, through all the centuries, been of immense comfort to the medical conscience. It soothes one's troubled feelings in all instances where we wish the patient had not died, and are a little uncertain of our relation to the final event. The old Greek's depressing remark comes to one's mind in the study of every case of acute mastoiditis. Yet experience will aid our judgment and is not necessarily fallacious, and the three indications I have mentioned are very safe guides in determining the question of choice between a continuance of antiphlogistic and sedative treatment, or of resort to trephining and free drainage.

THE FAUCIAL TONSILS: THE INDICATIONS FOR THEIR REMOVAL AND THE BEST METHODS BY WHICH TO ACCOMPLISH IT.

BY FRANCIS R. PACKARD, M.D.,

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The faucial tonsils, lying within the pillars of the fauces, are composed of lymphoid tissue with many follicles opening upon their surface, which is covered with mucous membrane lined with squamous epithelium. In early years the structure is soft and not very vascular. There is a normal tendency to their atrophy, manifesting itself usually at about the age of puberty. If they do not spontaneously atrophy at that time, they are liable to become the seat of pathological changes of varying severity as time goes on. The vascular supply during childhood is small; subsequently, as their structure becomes firmer, the blood-vessels increase in size and number.

The condition which ordinarily indicates removal of the faucial tonsils is that known as hypertrophy. This term should be applied exclusively to increase in size of the tonsils due to an over-development of their normal structure; all other pathological enlargements in size should be classed according to their underlying anatomical character. Several reasons, besides hypertrophy may render it necessary to remove the tonsils: (1) They may constitute a *locus minoris resistantiæ,* the openings in the crypts on the surface of the tonsil affording lodgement to various germs, especially those of diphtheria and scarlet fever. (2) Cyst formation is not infrequent, and is occasionally accompanied by the deposition of calcareous material. (3) There may be an accumulation of cheesy material in the crypts. (4) Tumors, principally sarcomata, are not uncommon.

As regards the etiology of hypertrophy of the faucial tonsils, but one fact can be considered as definitely established: it cer-

tainly most commonly occurs as an accompaniment of the lymphatic habit. Thus it is chiefly seen in children who show other symptoms of scrofulosis, enlarged glands, skin eruptions, etc.; or in rhachitic children, as in the famous case reported by Dupuytren, in which he attributed what is now universally conceded to have been a rhachitic deformity of the chest, to the influence of the obstruction to respiration produced by hypertrophied faucial tonsils. Occasionally parents will attribute the enlargement of the tonsils to an attack of scarlet fever or diphtheria, but it is more than likely that in such cases the acute disease has been the factor which called attention to the pathological condition in the throat.

The symptoms produced by simple hypertrophy of the faucial tonsils may be most striking. Almost invariably the enlargement is accompanied by the development of a mass of adenoid tissue in the nasopharynx. Examination readily reveals the prominence of the tonsils, but the facial aspect in itself is often sufficient to prepare one for the condition he will see upon examining the throat. The face in marked cases is that of the typical mouth-breather, because, although the obstruction is largely in the throat and not in the nose, the lack of air forces the child to open his mouth in order not to lose any air that he may possibly obtain. The child's stupid, heavy expression is apt to be borne out by its mentality and actions. Such children present the group of symptoms to which Guye, of Amsterdam, gave the name "aprosexia"; namely, loss of memory, deficient thinking power, inability to concentrate the mind, lack of intellectual force, and general mental apathy.

During the past winter the author has removed the tonsils from two children, one a boy and the other a girl, whose school-teachers had advised that they be kept from school and begin the next scholastic year in a class lower than that which they then occupied. In both instances, within a few weeks after the removal of their tonsils, the children had improved so markedly that they were able, with a little extra work, to resume their places in their classes, and they are now making the progress which they normally should.

Besides the above symptoms of respiratory obstruction, the child is apt to be restless at night, to suffer from dreams and nightmares, and to snore. Its bodily health suffers in consequence of this obstruction, and the result is that the child becomes puny, weak, and delicate. Other troubles may arise in the ears, the child gradually

losing its hearing, from obstruction of the Eustachian tube orifices. The digestion is often seriously impaired, and trifling ailments are apt to become serious, owing to the lack of resisting power of the child's organism.

Operative measures are the only really effective means of remedying hypertrophy of the tonsils; the application of chemical caustics or of salts of iodine is generally futile, and is more productive of harmful than of good results, as they irritate the tissues and result in the formation of inflammatory material, thereby making the mass hard and sometimes even stimulating it to more active growth. The use of the electric cautery is advocated by some operators, but it is tedious, and except in cases in which hemorrhage is to be feared does not possess very great value. I have used it in cases in which operation with the knife or other cutting instrument was declined. It requires that the patient should pay numerous visits to the office, usually fifteen or twenty sittings being necessary before enough punctures with the galvanocautery knife can be made to procure permanent reduction in the size of the tonsil mass. The rational procedure in practically all cases is to remove the offending tonsils with some variety of cutting instrument, but never while the tonsils are in an active state of inflammation. This removal should never be done in any person who is a "bleeder," since many fatal cases have been recorded following the operation in hæmophiliacs. In all cases careful inquiry should be made before operation as to whether there is any tendency to hemorrhage on the part of the patient. The operation is most usually performed in children, who may never have undergone any previous operative procedure which would be likely to attract attention to the existence of such a tendency, and unless the parents are closely questioned the operator runs a serious risk of involving himself in unpleasant consequences.

The cases readily divide themselves into very distinct classes,—those in which the patient is young, before the age of puberty, and those of more mature years. In children the tonsil tissue is soft and not very vascular, and the removal is not attended by very much subsequent pain; in adults the tissue is usually upon the seat of inflammatory troubles, there is considerable vascularity, and the tissues being more dense and harder to cut the subsequent pain is more severe.

Most operators prefer the tonsil guillotine, of which there are many varieties, to any other instrument for the removal of the tonsils. It was originated by Dr. Philip Syng Physick for removing redundant uvulæ, but was soon adapted by him for use on the tonsils. Physick's instrument consisted of a knife-blade sliding between two grooves on a steel plate; the idea was greatly improved upon by Fahnestock, who constructed one consisting of two steel rings which could be adjusted around the tonsil, and the concealed knife, which was in their circumference, was then drawn forward through it. At the present time the two varieties chiefly used are Maisonneuve's, or Matthieu's modification of it. This instrument is really a concealed ring-knife with a fork attachment, and is constructed in such a way that its use requires but one hand. The ring is slipped over the tonsil, the latter being secured by means of the fork at the end of a long shaft, and with the pushing motion the knife is carried neatly through the tissues, the excised piece remaining on the fork. The tonsillotome of Maisonneuve or Matthieu is so simple, so easily used, and so readily rendered aseptic, that it is by all means the most efficient instrument as yet devised for the removal of these organs.

The question of an anæsthetic is most important in the operation of tonsillotomy. Personally, I believe that whenever it is possible it is better to do the operation under local anæsthesia with cocaine; ether and chloroform are notoriously dangerous in operations upon the upper air-passages. It is frequently impossible to secure sufficient co-operation on the part of the patient, however, especially a child, to permit us to dispense with the administration of a general anæsthetic.

If I use a general anæsthetic I prefer ether; and in order to overcome the difficulty of lighting, I darken the room and use an electric head-light, thereby obviating all danger of an explosion. If ether is used the patient may be operated upon in an ordinary bed. He should be put thoroughly under the influence of ether, and then quickly raised and held upright by an assistant, who likewise steadies the head, clasping his hands over the vertex after bringing his arms through the patient's axillæ; another assistant or nurse should be at hand to hold the mouth-gag, hand instruments, etc. The removal takes but a few seconds. As soon as the patient is brought into the sitting posture, and the gag is properly inserted,

the operator throws his light into the mouth, places the tonsillotome quickly into position over the tonsil, and cuts it out. If possible he should not delay to check bleeding, but immediately remove the other tonsil. Immediately the operation is performed the patient should be returned to the recumbent position, lying on the abdomen with the head down in order that the blood may be prevented as far as possible from getting into the stomach and lungs. The mouth can be swabbed out with cotton on long applicators, styptics, such as Monsel's solution, rarely being required and generally being of but little service As soon as the patient recovers consciousness the mouth should be washed out with some cleansing antiseptic solution, and he should be allowed to suck cracked ice. For forty-eight hours after the operation only fluids or semisolids should be given, solid food being apt to impinge on the sore surfaces and irritate them.

If the operator has ascertained prior to the operation or during it that the patient has adenoids in the nasopharynx, as well as hypertrophy of the faucial tonsils, he should make an effort to remove the hypertrophied tissue from the nasopharynx at the time of operation. For this reason he should always be provided with either a Gottstein curette or some form of instrument for curettement of the nasopharynx.

In applying the tonsillotome the ring should be brought down firmly to the base of the tonsil, and it is of great importance to select an instrument of the proper size in each individual case; the commonest fault is the choice of one which is too large. Tonsillotomes can be bought in many sizes, and any surgeon who does many tonsillotomies should have at least three from which to make his choice. The ring of the tonsillotome may be procured in two shapes, one perfectly round and one oval. The author prefers the oval form, as in most instances it can be brought more perfectly into apposition to the surface of the tonsil.

During the operation there may be considerable bleeding, and a large quantity of the blood, in spite of all precautions to the contrary, is quite likely to be swallowed by the patient; this is usually brought up with the vomiting which results from the etherization. Cases have been reported in which a large amount of hemorrhage has occurred and has not been noticed until serious effects resulted, because of this disappearance of the blood within the stomach. Of

late a number of cases have been recorded of secondary hemorrhage following tonsillotomy, occurring on the third or fourth day after the operation. There have been very few, if any, deaths directly attributable to hemorrhage subsequent to removal of the faucial tonsils, except in instances in which the operation was performed in hæmophiliacs. In the average individual the hemorrhage checks of itself when enough blood has been lost to reduce the blood-pressure.

Following the operation the patient should be confined to bed for some hours and to his room for several days. The risk of infection of the open wound-surface is quite sufficient to render us very cautious about permitting his return to the bacteria-laden atmosphere of the streets.

The question frequently asked by patients or by the parents of children when it is proposed to do a tonsillotomy is the possibility of the return of the abnormal tissue. This practically never occurs. It is quite a common thing to see children who have had tonsillotomies performed upon them for the relief of obstructed respiration brought to one's office or to a hospital, the parents stating that relief has not been procured, and that they believe the removal of the tonsils was not complete or was followed by a recurrence of growth. In such cases it is usual to find that the operator has overlooked adenoid tissue in the nasopharynx. As stated above, the latter is a very common accompaniment of hypertrophied faucial tonsils, and removal of the latter alone, although it may give some relief, does not give nearly the required amount, provided there is any hypertrophied tissue whatever left in the nasopharynx. It should therefore be incumbent upon every operator to explore the nasopharynx with the finger upon completing the removal of the faucial tonsils, and thereby ascertain definitely whether or not any hypertrophied tissue is contained within it. If the removal of the faucial tonsils has not been complete, but some of their structure has been left, it is a disputed point as to whether the remainder will atrophy or will possibly grow still more and constitute a source of further trouble. It is my opinion that the removal of a portion of the tonsils usually results in the atrophy of the remainder, although I most firmly believe that the complete extirpation of the organs is the proper method of dealing with them.

Another question which is frequently asked by the parents is as

to the effect produced upon the voice by the removal of the faucial tonsils. This is especially so in instances where the patient is a choir-boy. It is true that many of the boys who possess most excellent choir voices are victims of enlargement of the tonsils, and among the laity there is a popular idea that this enlargement conduces to the excellence of the voice, and that the removal of the hypertrophied organs will result in commensurate diminution in voice power and beauty. In reality this is not so. The removal of the tonsils will possibly somewhat change the tone of the voice, having a tendency to obviate the falsetto which constitutes one of the prominent elements of boy voices, but as a rule it increases the volume and strength and adds to the purity of tone. This is a difficult question to decide, however, as frequently these boys make large sums of money, and it is rather a heavy responsibility, in case anything should go wrong with the boy's voice, to advise interference with it.

Special Articles

THE FUNCTION OF THE DIGESTIVE GLANDS, BASED ON THE RESEARCHES OF PAVLOF AND HIS PUPILS.[1]

A SPECIAL PAPER PREPARED AT THE REQUEST OF DOCTOR PAVLOF, PROFESSOR OF EXPERIMENTAL MEDICINE AT THE IMPERIAL INSTITUTE OF ST. PETERSBURG, RUSSIA.

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PART II.

THE PANCREAS.

The secretion of the pancreas is, generally speaking, subject to the same laws as the gastric secretion. Here also the quantity of juice poured out upon the food is proportional to the quantity of the food, and the character of the curve of the secretion is also peculiar to each variety of food, as is shown by the tables and by the curves (Figs. 1 and 2) of the secretion of the pancreatic juice.

One hundred grammes of meat, two hundred and fifty grammes of bread, and six hundred cubic centimetres of milk contain each the same amount of nitrogen. Of great interest is the comparison which Dr. Walter made in the determination of the quantity and quality of the work performed by the pancreas for each variety of food.

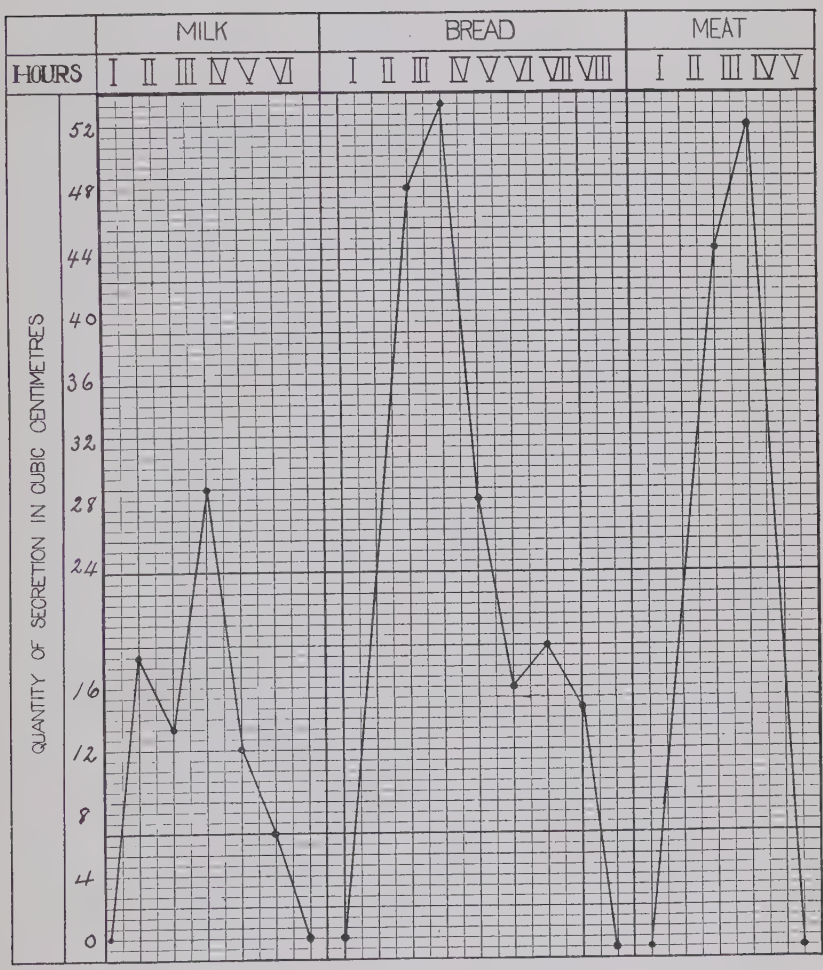
By concentration of the ferments he meant the square of the number of millimetres of the albumin or starch dissolved in tubes, or of cubic centimetres of alkali consumed during the titration of the forming acid; the time of the reaction (*cf.* above) being proportional, according to the law of Schütze and Borissof, to the

[1] Translated from the Russian by Robert Formad, M.D.

TABLE SHOWING THE QUANTITY AND THE QUALITY OF THE PANCREATIC SECRETION BY HOURS.

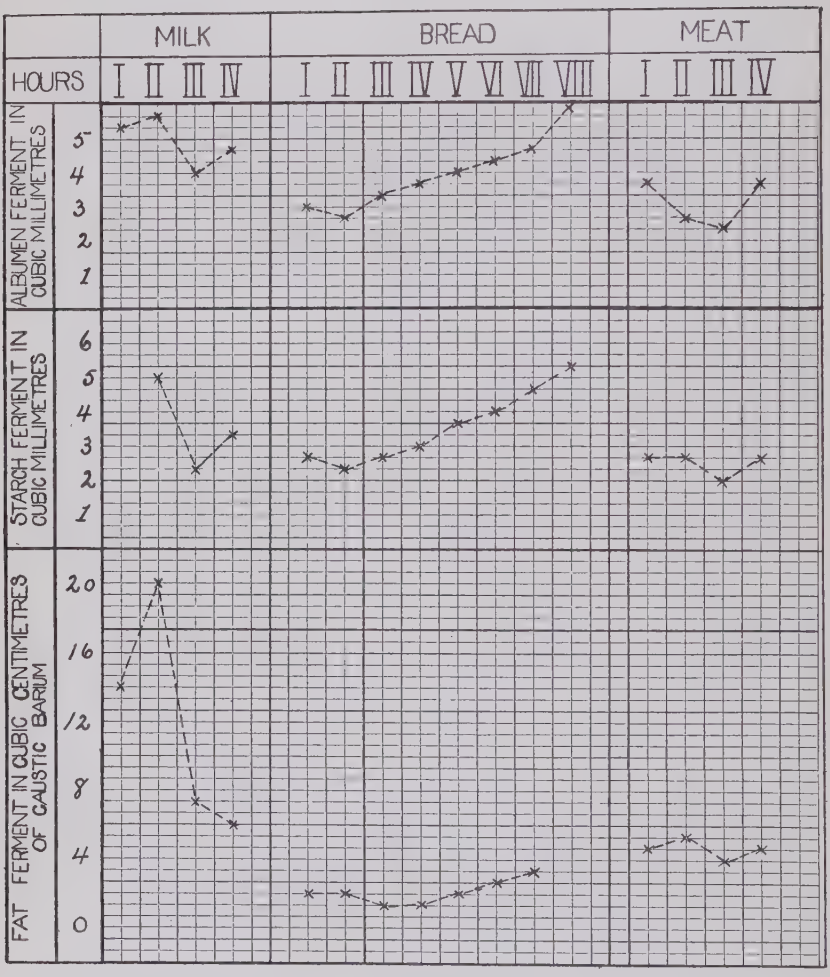
Hours.	From 600 Cubic Centimetres Milk.	From 250 Grammes Bread.	From 100 Grammes Meat.	The Digestive Power of the Individual Pancreatic Ferments.								
				600 Cubic Centimetres Milk.			250 Grammes Bread.			100 Grammes Meat.		
				Albumin Ferment.	Starch Ferment.	Fat Ferment.	Albumin Ferment.	Starch Ferment.	Fat Ferment.	Albumin Ferment.	Starch Ferment.	Fat Ferment.
	C.c.	C.c.	C.c.	Mm.	Mm.	C.c.	Mm.	Mm.	C.c.	Mm.	Mm.	C.c.
1	8 5	36.5	38.75	5.75	5.0	14.3	3.0	2.75	2.2	3.5	2.62	5.2
2	7.6	50.2	44.6	5.88	5.0	19.7	2.88	2.38	2.1	2.88	2.5	5.7
3	14.6	20.9	30.4	4.25	2.38	7.0	3.5	2.62	1.6	2.5	2.0	4.1
4	11.2	14.1	16.9	4.5	3.31	5.9	3.88	3.12	1.7	3.88	2.69	4.8
5	3.2	16.4	0.8	....	....	....	4.12	3.88	2.1	....	....	....
6	1.0	12.7	....	....	....	....	4.25	4.25	2.5	....	....	....
7	1.0	10.7	....	....	....	....	4.62	4.75	3.1	....	....	....
8	1.0	6.9	....	....	....	....	6.0	5.12	....	....	....	....

FIG. 1.



Comparison of pancreatic secretion for milk, bread, and meat expressed in cubic centimetres.

FIG. 2.


MILK
BREAD
MEAT
HOURS
I II III IV
I II III IV V VI VII VIII
I II III IV
ALBUMEN FERMENT IN CUBIC MILLIMETRES
5 4 3 2 1
STARCH FERMENT IN CUBIC MILLIMETRES
6 5 4 3 2 1
FAT FERMENT IN CUBIC CENTIMETRES OF CAUSTIC BARIUM
20 16 12 8 4 0

Curves showing action of the various pancreatic ferments on milk, bread, and meat.

square of the percentage of the ferment present in the digestive fluids. By the absolute number he meant the product of that square by the number of cubic centimetres of the secreted juice.

Food.	Quantity of juice in c.c.	Albuminous Ferment.		Starchy Ferment.		Fatty Ferment.	
		Concentration of the ferment.	Absolute number of ferment units.	Concentration of the ferment.	Absolute number of ferment units.	Concentration of the ferment.	Absolute number of ferment units.
600 c.c. milk	48	22.6	1085	9	432	90.3	4334
250 grammes bread	151	13.1	1978	10.6	1601	5.3	800
100 grammes meat	144	10.6	1502	4.5	648	25.0	3600

Considering the given tables and curves, we see that each kind of food is acted upon by a corresponding quantity and a corresponding quality of juice, and that the character of the secretion is peculiar to each particular kind of food. The most concentrated in regard to the albuminous ferment appears to be milk, and next in their order are found bread and meat. In regard to the starchy ferment bread stands first, then milk and meat. And finally, in regard to the fatty ferments, the first position seems to be assigned to milk, followed next by meat and then by bread. That the richness of the milk is well adapted for the fatty ferments, and of the bread for the starchy ferments, appears so evident as to be beyond doubt. It may seem strange that the concentration of the albuminous ferments is highest in a milk diet, while the gastric juice's action on milk is weaker than for other foods. That this involves no contradiction, however, is best seen if we consider not the concentration, but the general quantity (the absolute number) of the ferments secreted by the glands. As a matter of fact, the greatest quantity of albuminous ferment is secreted on bread (1978 units), less on meat (1502), and least of all on milk (1085). The greatest amount of fatty ferment is secreted on milk, and of starchy ferment on bread.

Very interesting, both from a theoretical and a practical point of view, is the following fact. A dog was kept on a bread-and-milk diet for a long while (one and one-half months) and then was put on a meat diet. The increase in the strength of the albuminous ferment and the decrease in that of the starchy ferment did not appear at once, but only after a long interval of time, which shows that when the gland becomes adapted to a certain kind of action, it experiences some difficulty in changing to a new one and must take time to do so. The same results were obtained, in general, when the dog was kept for a long time on a meat diet and then transferred to a milk-and-bread diet. Thus, at the outset the digestive force exerted upon the albumin was expressed by four millimetres of the digested albumin; after ten days of the milk-bread regimen it was 2.25 millimetres; after twenty-two days, 1.25 millimetres; after twenty-four days more it was equal to zero. All the above data clearly indicate that the pancreas in some way finds out what sort of food enters the intestine, and correspondingly prepares and pours out into the latter its juice.

Now, the question is, How does the pancreas recognize the sort of food which enters into the intestine? While studying the activity of the stomach, we have seen that it is the mucous membrane that recognizes the food. Here we may expect something similar. And, in fact, the experiments on the secretion of the pancreas have not only fully confirmed this, but have, moreover, widened our conception of the specific irritability of the mucous membrane of the gastro-intestinal tract and of the reflexes emanating therefrom. We have seen above that the fat introduced into a dog's stomach (or, still better, directly into the duodenum) retards the secretion of the gastric juice. We must add that it also considerably increases the secretion of the pancreatic juice. Thus we see that the mucous membrane of the duodenum, recognizing the presence of fat in the intestine, sends out at once two messages, one ordering the retarding of the gastric secretion and the other the increasing of the secretion of the pancreatic juice.

The passing of the acid contents of the stomach into the duodenum is of great importance for the secretion of the pancreatic juice. If a 0.5 per cent. solution of muriatic acid is poured into the stomach of a dog, or directly into his duodenum, there appears within two or three minutes the secretion of the pancreatic juice.

The sensitive part that recognizes the presence of the acid appears to be the mucous membrane of the duodenum, not that of the stomach; for if the acid is introduced into the stomach, with the passage into the duodenum tied up, no secretion of the pancreatic juice takes place; but it does take place directly on introducing this acid, in the same experiment, into the duodenum.

That the acid entering the duodenum incites the pancreatic juice in a reflex manner by irritating the mucous membrane, and not by absorption, can be shown experimentally by introducing the same acid into the rectum; these experiments always gave negative results as to the secretion of the pancreatic juice, even upon introducing into the rectum much greater quantities of acid. Other acids, as phosphorous, citric, lactic, or acetic acid, also increase or stimulate the secretion of the pancreatic juice.

The concentration of the acid introduced greatly influences the quantity of the secreted pancreatic juice. Thus, on introducing into the stomach 250 cubic centimetres of .5 per cent. solution of hydrochloric acid, there were secreted from 70.8 to 89.4 cubic centimetres of pancreatic juice. On the introduction, however, of a .1 per cent. solution, the secretion was only 25.7 to 32.5 cubic centimetres; and on the introduction of a .05 per cent. solution, only 20.5 cubic centimetres.

Of further interest is the following fact. On the introduction of the acid into the stomach, the secreting pancreatic juice appears to be more alkaline than in the case of any other food, yet much poorer in organic substance. Thus we have here a high degree of adaptability in the function of the pancreas. Since the hydrochloric acid and the gastric juice stimulate the pancreas, it was natural that, in experiments seeking to discover whether the activity of the pancreas is stimulated directly by the given agent, the influence of the secreting gastric juice should not escape attention. To exclude this latter influence, the experiments were performed on dogs which had not only a fistula of the pancreas, but also one of the stomach, an opportunity being thus obtained to observe the state of the stomach. When olive oil was introduced through the fistula into the stomach, it was noticed, after from two to three minutes, that a considerable secretion of the pancreatic juice took place (from seven to fifteen cubic centimetres in fifteen minutes); when the fistula of the stomach was opened, in from fifteen to thirty

minutes, the stomach contained, in addition to the oil, only from three to five cubic centimetres of alkaline mucus. On introducing into the stomach one hundred and fifty cubic centimetres of water, Dr. Damaskin noticed, after two or three minutes, either the appearance of the pancreatic juice or a considerably increased quantity of the secretion, if any had taken place before; and when, within one or two minutes, he removed the fluid from the stomach, the latter proved to be alkaline. Similar positive results were obtained on introducing a solution of Liebig's extract. But starch paste, as a promoter of the gastric juice, gave a negative result.

We have already indicated, while examining the causes which stimulate the secretion of the gastric juice, the peculiar action of starch,—namely, that in itself it does not cause the activity of the gastric juice, but when added to meat it changes the composition of the former. Here we have the same thing.

From the above tables of the secretion of the gastric and pancreatic juices, it can be seen that in the action of the different substances upon the mucous membrane of the gastro-intestinal tract, as well as in the secretion of the juices, two distinct factors must be taken into consideration,—namely, the quantity and the quality of the juices. This is noticed, with particular emphasis, in the experiments of Dr. Walter, conducted with the object of making clear the influence of fat in milk. On introducing through the fistula six hundred cubic centimetres of milk filtrate—milk freed from fat by filtration—into the stomach of the dog, the secreting pancreatic juice was found to contain from 5.1 to 6.5 conventional units of fatty ferment; while on introducing a mixture of five hundred and seventy cubic centimetres of milk filtrate plus thirty cubic centimetres of almond oil, the secreting pancreatic juice was found to contain from 11.3 to 13.7 units of fatty ferment; and on introducing one hundred cubic centimetres of olive oil, the pancreatic juice was found to have, in fifteen minutes, 15.5 units of fatty ferment. Thus, the connection between the fatty irritant and the function of the juice as a ferment is quite apparent. The quantity of the secreting juice has no bearing upon its richness in this or the other ferment.

Having thus established the importance of hydrochloric acid and the principal component parts of our food, it now remains for us to make clear what influence the mind has upon the secretion of

the pancreatic juice,—in other words, to point out whether there is a psychical secretion. If to a dog upon which œsophagotomy has been performed, and which has, besides, a fistula of the stomach and a fistula of the pancreas, we administer fictitious feeding, then, although there will be a secretion of both gastric and pancreatic juices, a difference will be noticed in the time of the appearance of the secretion of both juices, the gastric secretion not occurring until four and one-half or five minutes after the beginning of the feeding, and the pancreatic secretion appearing after two or three minutes. For this reason we can admit the existence of a psychical secretion also in the pancreas. However, seeing that the secretion of the psycho-gastric juice promotes the secretion of the pancreatic juice, we must consider the secretion of the psychical pancreatic juice to be only of secondary importance.

Having before us a function so complex and harmonious, let us now examine the mechanism of innervation which governs the action of the pancreas. Before special investigations were made in the laboratory of Pavlof our knowledge as to the importance of nervous mechanism for the activity of the pancreas was disconnected. From the researches of Heidenhain and Landau, it was known that irritation of the medulla oblongata stimulates, in certain cases, the pancreas. Then Bernstein, Pavlof, and Afanasyef showed that irritation of the sensory nerves causes the retardation of the secretion.

By the investigations made in Pavlof's laboratory, two nerves were proved to be the secretory nerves of the pancreas,—namely, the vagus and the sympathetic. To discover the presence of the secretory fibres for the pancreas in these nerves was, however, not an easy matter, and attempts made by previous investigators were without results. There are two reasons for this, the first being the extreme sensitiveness of the pancreatic circulation or blood supply, the second, the existence in these nerves besides secretory fibres of other fibres retarding the activity of the gland.

To show the secretory effect of the irritation of the vagus it is necessary to conduct the experiment with some precautions. Tracheotomy is very carefully performed upon a normal animal, the spinal cord is quickly severed under the medulla oblongata, and artificial respiration is introduced. Then the opening of the thorax is proceeded with more slowly, in order to locate the vagus nerve,

and the opening of the abdomen, for the purpose of inserting a canula into the duct of the pancreas. The quick and, as far as possible, painless operation at the beginning, before the severing of the spinal cord, is necessary in order to exclude the harmful, reflexively retarding influence of the vagus nerve. The irritation of the vagus above the diaphragm is for the purpose of excluding the retarding influence of this nerve upon the heart. Even with such precautions for obtaining a permanent secretory effect of the vagus, it is necessary for a while to irritate it repeatedly without visible results, but later there appears a secretion of juice at every irritation.

If by the irritation of one vagus we obtain a permanent secretory effect and to this add the irritation of the other vagus, we notice a retardation of the secretion. Still more distinct was the retardation of the vagus observed in the experiments of Dr. Popelski (in the Pavlof laboratory). He discovered special branches of the vagus which were specifically concerned in retardation. In a rapid experiment this retardation can be shown in the following manner. By introducing into the duodenum a solution of hydrochloric acid an abundant secretion of pancreatic juice is obtained. If then we apply the irritation to the vagus or to the above-mentioned retarding branch of it, there will be noticed at once either a decrease or a complete cessation of the secretion of the pancreatic juice. To show the secretory influence of the sympathetic nerve, containing vasoconstrictor fibres, a method must be found which will exclude the vasoconstrictor action. For that purpose the sympathetic nerve is irritated either by means of Heidenhain's tetanometer or by an electric current, not immediately after the operation, but four or five days after the severance of the nerve.

THE PASSAGE OF THE FOOD FROM THE STOMACH INTO THE DUODENUM.

As indicated above, the chief *rôle* in the secretion of the pancreatic juice is played by the mucous membrane of the duodenum, which recognizes acids and fat; and hence, in order to obtain a clear conception of the activity of the pancreas, as well as of the activity of other glands, as the liver and Lieberkühn's glands, we must first of all know the main principles which govern the passage of food from the stomach into the small intestines.

Dr. Serdyukof, introducing through the fistula into the stomach water, a .5 per cent. solution of carbonate of sodium, and a .5 per cent. solution of hydrochloric acid, and opening it again at different times, found, in conformity with the investigations of Hirsch, that the passage of the contents of the stomach into the intestines is determined by both the stomach and the duodenum, the acid irritating the mucous membrane of the intestine closing the pylorus reflexively. This shows clearly that the more acidulated the contents of the stomach are, and the less pancreatic juice is secreted in a unit of time, for the neutralization of the acid entering from the stomach into the intestines, the slower will be the passage of the contents of the stomach into the intestines, and *vice versa.*

We thus see that the passage of the food from the stomach into the intestines is regulated by the sensitiveness of the mucous membrane of the duodenum. The experiments of Dr. Lintvarof, performed upon two dogs, one of which had two fistulæ of the stomach, one of which was near the pylorus, and the other a fistula of the stomach and the duodenum, show that the first effect of the hydrochloric acid after its escape from the stomach is to increase the reflex activity of the pylorus by its action upon the duodenal mucous membrane. This hyperactivity diminishes, however, with equal rapidity upon the acid becoming neutralized by means of the alkaline intestinal juices. Besides the acid, the same retarding influence upon the exit of food from the stomach is also exerted by fat introduced into the duodenum; the reflex of the fat in this case, however, though appearing just as quickly, disappears very slowly, lasting from one to three hours. The stenosis of the pyloric ring is also produced by soaps (0.2 to 0.5 per cent.), by lactic acid, and in a less degree by glycerin, peptones, and beef extract. The object of the slow passage of the gastric contents into the intestines is clear. It is to give an opportunity to the intestinal juices, the pancreatic secretion, and the bile, to act upon the passing food.

In this connection, the experiments of Dr. Zhegalof are of interest. He observed the secreting action of the stomach while the duct of the pancreas was ligatured. He drew his conclusions as to the secreting action of the stomach from the secretion of a small isolated stomach. From the above we might expect that the tying up of the duct of the pancreas would impede the passage of food from the stomach into the intestines, especially foods containing fat, and

that with the retaining of food in the stomach the irritation of the gastric glands must increase. And if we recall the above-mentioned fact of the retarded secretion of the gastric juice in case of its accumulation in the stomach, it will be easily perceived why the gastric secretion while the duct of the pancreas is tied up will not be even and continuous, but intermittent; that is, as soon as the acid disappears from the stomach a new secretion takes place. Indeed, the scheme of the secretion of the gastric juice and of the passage of food from the stomach into the intestines fully meets our expectations. The gastric secretion while the duct is tied up is considerably slackened, and is characterized by its wave-like rise and fall, its interruptions, and even complete cessation, during the presence of food in the stomach. The deviation of the secretion was greatest in fatty foods (milk and yolks of hens' eggs), less in bread, and least of all in meat. It is of interest to note that in the experiment of feeding on bread (two hundred grammes) Dr. Zhegalof found that at the end of the period for the secretion of juices in the stomach the remaining bread mass, on being dried up with a blotter, weighed, after all, from forty to one hundred and ten grammes. A similar retaining of food in the stomach was also noticed in feeding with yolks of eggs. Considering, in the first place, the fact that food is retained in the stomach while the duct of the pancreas is tied, and, in the second place, the part played by the pancreatic juice and the digestion of these foods, we cannot help seeing the great adaptability found in the organism, not only with regard to the secretion of the juices, but also with respect to the conveyance of food through the gastro-intestinal tract.

THE FLOW OF THE BILE INTO THE DUODENUM.

The bile, a result of life activity of the walls of the hepatic cells, may be regarded as a product of excretion as well as a product of secretion of the organism. The excretory function of the liver can best be observed through a fistula of the gall-bladder. The numerous observations which were thus made show that the bile is secreted constantly, whereas every acceleration in the current of the portal vein causes a like acceleration in the current of the bile and its various constituents. The intimate and harmonious relation between the bile secretion and the rapidity of circulation of blood in the liver are perfectly clear when we bear in mind that the object

of this process is to carry off injurious substances from the organ. We also see a perfect analogy to the functions of the kidneys. Whenever the digestive function begins in the gastro-intestinal tract, the circulation in the portal vein is increased, and this collects the blood from the whole tract; together with this there is an increase in the formation of bile. The latter should be regarded as a process of excretion from the liver. From the discoveries of Schwahn we learn that the bile empties into the intestinal tract, becoming at the same time a useful secretion. Many investigators have pointed out that the bile checks the gastric secretion and stimulates the secretion of the pancreas, facilitating the digestion and absorption of fat. The action of bile in aiding the digestion of fat, albumin, and starch has been confirmed in Pavlof's laboratory, Dr. Zhegalof observing a small quantity of diastatic and proteolytic ferment in the biliary secretion. When the bile is once formed, it is not only an excretion but also a secretion,—that is, a fluid which is useful in the bodily economy.

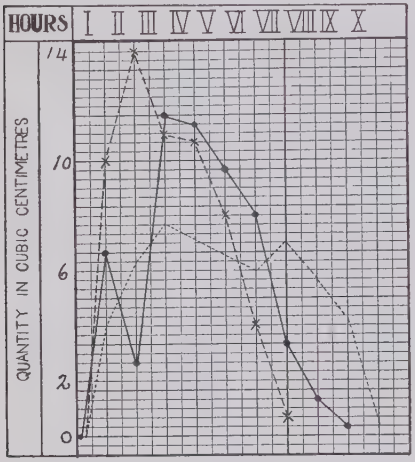
Remembering the tendency to adapt the digestive juices to the quantity and quality of food, it might be expected that the secretion of bile in the intestinal tract would also be regulated by certain conditions, and that the bile would not flow constantly but only when it is needed. To observe the flow of bile we must have dogs with an external opening of the ductus choledochus rather than with an ordinary opening into the gall-bladder. Experiments along this line by Drs. Bryuno and Klodnitzky prove the same adaptation of bile flow as was found in connection with other digestive juices. The following figures show the secretion of bile in the duodenum when a dog was given varying foods containing the same amount of nitrogen.

Hours.	600 c.c. milk.	100 gms. meat.	250 gms. bread.	Hours.	600 c.c. milk	100 gms. meat.	250 gms. bread.
1	7.5	9.2	3.7	6	7.5	3.7	6.2
2	3.7	14.7	7.1	7	3.7	0.6	6.5
3	13.6	12.6	7.8	8	1.8	——	5.3
4	12.9	11.4	7.2	9	0.8	——	3.9
5	9.5	8.0	6.5	10	——	——	1.5

By this record we see that the largest quantity of bile during milk diet was formed in the third and the fourth hour, during meat diet in the second hour, and during bread diet the quantity reached and maintained a certain level and afterwards fell gradually. It

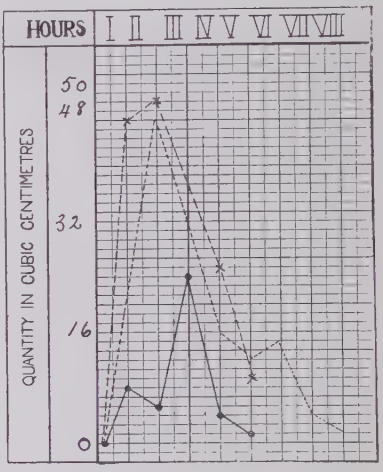
has been stated that the bile increases the digestive power of the pancreatic juice quite perceptibly, and the following curves (Figs. 3 and 4) record the pancreatic secretion in connection with various foods during the flow of bile into the intestine.

FIG. 3.



Record showing secretion of bile.

FIG. 4.



Record showing secretion of bile.

(The continuous line shows the curve of secretion following the ingestion of six hundred cubic centimetres of milk; the dark line, of one hundred grammes of meat; and the dotted line, of two hundred and fifty grammes of bread.)

There is a noticeable correspondence between the flow of bile and the pancreatic secretion, and the significance of this condition is clear. As to the quantity of bile under the influence of various foods with the same quantity of nitrogen, Dr. Klodnitzky secured the following average in experiments upon two dogs:

	First dog.	Second dog.
600 c.c. milk	61.3 c.c.	37.5 c.c.
100 grammes meat	61.0	37.8
250 grammes bread	55.7	34.9

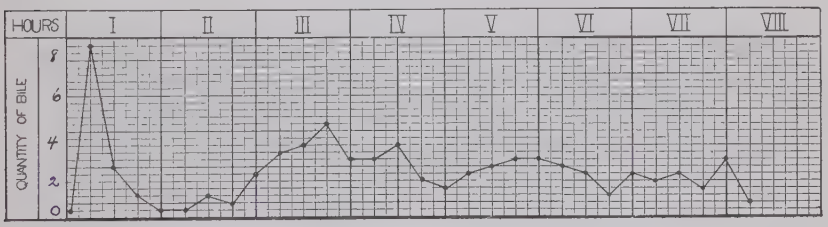
When one-half the quantity of food was given, about one-half the amount of secretion was obtained. The noticeable difference as between the two dogs may be partly accounted for by the fact that the first dog partook of the food greedily while the second was rather indifferent. If we take into consideration the plentiful flow of bile during meat and milk ingestion and the moderate flow during bread ingestion, we can surmise that the difference in bile secretion between the two dogs was due to the character of food

previously eaten; the dog which secreted a small amount of bile was probably used to an exclusively vegetable diet.

The above curves show that some time elapses between the moment of contact of the food with the stomach and the beginning of the flow of bile. According to the investigations of Dr. Klodnitzky, in milk diet the time is about twenty minutes, in meat diet thirty-six minutes, and in the case of bread it is forty-seven minutes. The length of this period and its variation with several foods suggests (1) that the psychical excitement does not have any influence upon the flow of bile into the intestine, and (2) that the mere presence of food in the stomach does not cause this flow.

In order to show more clearly the character of the bile flow, we present a curve which indicates the condition in quarter-hour periods. (Fig. 5.)

FIG. 5.



A study of the facts as elicited leads to the suggestion that the flow of bile may be dependent upon the passage of the food from the stomach into the duodenum. By means of a glass tube inserted into an intestinal fistula the amount of food that passed from the stomach was noted, at the same time that the bile flow was measured. The passage of milk from the stomach consumed an average of thirteen minutes from the time of its ingestion, while the flow of bile appeared within five minutes after the passage of the food into the intestine. When meat was given, the passage consumed fifteen minutes, while the bile flow began twenty-one minutes after the passage of the food into the intestines; in the case of bread ingestion the periods were respectively nineteen minutes and twenty-six minutes. The prolonged interval in meat and bread ingestion suggests that some transformation in the food may be necessary before the bile flow commences. Dr. Klodnitzky secured similar results by administering fifty grammes of castor oil and by the use of water and five-tenths per cent. solution of sodium nitrate, proving that these did

not promote bile flow. In nine experiments Liebig's extract gave positive results only twice. Egg-albumin when placed directly in the stomach did not promote bile secretion, but when eaten did so. The products of albumin and fibrin digestion likewise stimulated this secretion, while fat, in the shape of almond oil, olive oil, and butter, appears to be the most active bile promoter.

When fat or milk was placed directly into the duodenum, it was repulsed into the stomach. In order to retain the milk poured into the duodenum Dr. Klodnitzky was obliged first to tease the dog with the milk and then introduce small quantities at a time. When this was done, the secretion of the pancreatic juice was promoted and the closure of the gastric exit was secured. In these cases the flow of bile always began within three or four minutes, and he observed some connection between the peristaltic action of the intestine and the bile flow.

THE INTESTINAL JUICE.

Although Thiry's operation, performed as early as 1864, enabled us to obtain pure intestinal juice, until very recently we had no positive data to determine its function. Some believed that it contained a ferment for digesting albumin; others thought that it was for the digestion of starch and cane sugar; still others denied the presence of either ferment. Dr. Shepovalnikof in Pavlof's laboratory succeeded at last in determining the real function of the intestinal juice in digestion. He discovered in it a specific ferment, called "chymaze," the main function of which is to stimulate the activity of the ferments of the pancreatic juice. By adding unboiled intestinal juice to the pancreatic juice we usually obtain an accelerated digestion of albumin, starch, and fat, but on adding boiled intestinal juice no acceleration in digestion is noticeable. The intestinal juice by itself has a very weak independent action on starch, a doubtful one on fibrin, and no effect at all on the white of hens' eggs. The most beautiful experiments are those with pancreatic juice which is very weak in its action or does not at all digest the white of a hen's egg or fibrin. Such pancreatic juice can be obtained in rapid experiments soon after the making of a pancreatic fistula, and also after a prolonged feeding of dogs on carbohydrated fatty food. In such experiments—say, on the digestion of the white of a hen's egg in glass tubes—we obtain zero digestion with

the pancreatic juice alone, zero digestion with the intestinal juice only, but about five or six millimetres with a mixture of both. In the digestion of fibrin in such cases we notice a decrease in the time of digestion from hours to minutes.

With the discovery of this chymaze it became possible to enter more deeply into the chemical processes of digestion which take place in the intestinal tract.

We have seen above that after a prolonged bread-and milk diet there is produced in dogs a pancreatic juice very poor in albuminous ferment, but that a very strong albuminous ferment is obtained after a long-continued diet of meat. The addition of the intestinal juice, chymaze, to one of the other juices shows that a marked acceleration of the digestion of albumin takes place in the juice only after a bread-and-milk diet, while the addition of chymaze to the pancreatic juice after a prolonged meat diet is either without any effect on the time of digestion or even slightly retards it. These experiments thus prove that the pancreatic juice differs in the albuminous diet from that in the bread-and-milk diet: in the first it appears to be in an unaltered state, and in the second in the form of a zymogen. But on the discovery of this phenomenon the question arises, What is the object of the organism in producing albuminous ferment in the form of zymogen in the bread-and-milk diet? The experiments to determine the changes in strength of the fatty and starchy ferment have demonstrated that when the pancreatic juice contains the albuminous ferment in an unaltered state and also in the state of zymogen, then in the unaltered state the albuminous ferment destroys the starchy and fatty ferments very rapidly. Hence there is no doubt that in a bread-and-milk diet it is of greater advantage to the organism to secrete pancreatic juice with the albuminous ferment in a zymogenous state, which will show its effects only some time after it has been acted upon by the intestinal juice. Shepovalnikof's experiments show that it requires about thirty minutes for the intestinal juice fully to produce its stimulating effect upon the pancreatic juice.

Having thus shown the function of the intestinal juice, we will now proceed to make clear the conditions of its secretion. The observations of the secretion of the intestinal juice on an isolated section of the intestine have shown that before we introduce a tube into the intestine, which is a mechanical irritant, the secretion is

insignificant, uneven, occurs at long intervals, and is often entirely absent; but on the introduction of a tube (usually one of rubber) the secretion begins immediately, irrespective of whether the dog was with or without food. Hence it is clear that one of the conditions of secretion is mechanical irritation. Subsequent experiments with the introduction into an isolated portion of the intestine of different foods for a given short time (ten seconds), and the observation during the exit of those foods of the quantity of chymaze secreted in the intestinal juice, have shown that the pancreatic juice is the only specific stimulant for the secretion of the chymaze. The introduction of the former even in minute quantities increases the contents of the chymaze in the intestinal juice to a marked extent. The advantage of this phenomenon is self-evident.

In conclusion, having made clear the scheme of the secretion of the different juices, we will dwell again, in a general way, on the psychical influence on digestion. For this purpose we shall examine the effects on the secretion of the juices by the severing of the vagus, resulting, as we have seen, in the disappearance of the secretion of the psychical juice in the stomach. On account of the absence of the psychical juice in the stomach, the secretion of the gastric juice through chemical irritants also becomes difficult, since we know that often it is not the substances which are introduced that act as irritants, but their products. The gastric juice acts as a stimulant to the secretion of the pancreatic juice, so that we notice in this case also the diminution of the secretion of the pancreatic juice; and the insufficiency of the gastric and pancreatic juices will cause a retardation of the food in the stomach, followed by its decomposition. The insufficiency of the pancreatic juice will also be reflected on the secretion of the intestinal juice, as well as on that of the bile, since the stimulants for the discharging of the bile into the duodenum are also not the substances introduced, but their products digested by the gastric and pancreatic juices.

Whatever has been said above concerning the secretion of the digestive juices is evidently also of great clinical importance. This is very clearly illustrated by the following clinical case observed by the author.

A woman, who, by reason of a cancerous stenosis of the œsophagus, was fed on eggs and milk, through a fistula made in her stomach, developed a marked disorder of the gastro-intestinal tract

(diarrhœa), and, as a result, was losing daily in weight. At a consultation the author pointed out, first, that under the given method of feeding,—that is, the pouring of food into the stomach,—there was a complete absence of the secretion of psychical juice, and, second, that the milk-and-egg food lacked strong stimulants for the secretion of chemical juice. The small quantity of gastric juice was followed by the retention of the food in the stomach, as well as by its decomposition. This also caused the diminution in the secretion of the pancreatic juice, bile, and intestinal juice; all of which combined to produce not the assimilation but the decomposition of the food in the intestines. On these grounds, in order to stimulate the secretion of the psychical juice, the author suggested that the patient chew her favorite food and then remove it from the mouth; further, as this chewing is not equivalent to normal eating as a psychical stimulant, he proposed that one hundred cubic centimetres of normal gastric juice obtained from an œsophagotomied dog should be introduced into her stomach together with the food; and, finally, that a cup of strong beef tea should be poured in to stimulate the secretion of the gastric juice by chemical irritation. The food remained the same,—milk and eggs. Through the application of this method of feeding the disordered activity of the intestines rapidly disappeared, and, despite the progress of the cancer, which caused her death a month later, owing to the rupture of a large vessel and subsequent hemorrhage, the patient during that time gained four kilogrammes in weight.

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A CRITICAL STUDY OF THE THEORY OF INFLAMMATION.

FROM A LECTURE BEFORE THE SOCIETY OF GYNÆCOLOGY IN MUNICH.

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Gentlemen,—In a discussion on definitions by John Stuart Mill, the English philosopher, there is contained the following passage, which I quote briefly, according to its substance: "Language is not made, but grows, as Sir James Mackintosh said of governments. A name is not given to a class of objects all at once and arbitrarily, but is applied first to one object and then, by a series of transitions, to another, and then another, until finally it embraces things that have nothing in common with those to which it was originally applied. As it is still retained by them, however, it happens that the name comes to designate in the end a confusion of objects which have nothing in common and indicates only a vague and general similarity. When it has reached this stage, it has become useless as a medium of thought and of communicating thought; its utility can be restored only when it divests itself of part of its diversity and is limited to objects which have certain attributes in common. These are the disadvantages of a language 'which is not made, but which grows.'"

With such disadvantages we are often confronted in medical language. We know how often in autopsies or in the examination of excised specimens the pathologist is asked by the practitioner whether or not he considers a certain change to be inflammatory, and how often this question is answered with as little precision as it is variously construed by the questioner. Often enough, we must admit, both parties, after a longer or shorter discussion, part company without obtaining satisfaction. In special branches it not seldom happens that the clinician determines his own terminology as regards anatomical questions, without concerning himself in the least as to the anatomical bases of the disease. On the other hand,

you will search in vain even in the text-books of pathological anatomy for a uniform definition of inflammation, and it requires no extensive knowledge of literature to become convinced that the confusion of which Mill speaks is most adequately exemplified in the chapter on inflammation.

Let us, therefore, follow the path indicated to us by the logician, and find the original meaning of the word inflammation; we shall then determine to what manifestations this name can be applied according to our present state of knowledge. Originally the name was used to indicate a purely clinical symptom complex, as seen from direct observation of external, visible parts, and consisting of the four well-known *cardinal symptoms* of Galen: *rubor, tumor, calor,* and *dolor.* The word inflammation (L. *inflammatio*), then, is but an incomplete, figurative designation of a group of manifestations which might with equal propriety have been designated "burn," if the word had not already been employed in another sense. Judging mostly from analogy, phenomena similar to those observed in external, visible parts were assumed also for certain diseases of the *internal* organs, especially when accompanied by a feverish rise in temperature, a general phenomenon apparently closely related to the local symptom complex of inflammation. It is true that here the application of the name no longer depends upon purely direct observation, since in internal organs this is restricted almost exclusively to the results of autopsy; naturally, however, these can only afford incomplete pictures of the diseased condition in the living state and at best only make possible a recognition of redness and swelling.

It was natural that in their investigations scientists would in the long run not rest satisfied with the establishment of a purely external symptom group, but following a causal necessity would seek to learn upon what the above-mentioned manifestations depended, what were the anatomical processes underlying them; in other words, would attempt an anatomical analysis of the general process known as inflammation. Of the results obtained, I need only remind you here for the sake of completeness. It showed the presence of active hyperæmia, increased plethora of the inflamed area, differing from simple congestion in duration, and accompanied by filling and dilatation of the blood-vessels, as well as by acceleration and subsequent slowing of the blood-current, even to complete

stasis, and by the peripheral drift of the leucocytes; there occurs further an increase in the physiological transudation, in the passing of fluid components of blood from the vascular system, combined with a change in the chemical character of the outcoming liquid, which is decidedly richer in albumin than the physiological exudate and not seldom coagulated; finally, the microscopic examination of inflamed organs proved that the inflammatory exudate is largely admixed with cells, which, coming from the blood, have emigrated through the cell-walls, and under certain circumstances issue in such great masses that the exudate acquires a distinctly cellular, purulent character. The above processes afford an explanation of the principal clinical manifestations. The redness of inflammation is to be explained by the congestive hyperæmia, as is also partly the temperature of inflammation; the accumulation of exudates in the tissue causes the inflammatory tumor, or swelling; the excretion of exudates on surfaces gives rise to inflammatory secretion; the inflammatory pain, which is the least constant symptom of all, is easily explained by the irritation of the nerve endings.

By this anatomical analysis of the inflammatory symptoms we have gained one step,—we have found a common point around which all the phenomena hitherto considered must be grouped: the vascular apparatus. If, therefore, the inflammatory vascular alteration is designated as the characterizing factor, this does not by any means explain the process, for, strictly speaking, the expression merely indicates a lacuna in our knowledge which yet remains to be filled; nevertheless, we have, as Roux said, put the interrogation mark in the right place and thus gained the advantage of knowing what is the real question. There must have been good grounds for departing from this theory of inflammation, which in itself is so very clear, and wandering forth in quest of a conception based on different principles; but the path of anatomical investigation once entered seemed to lead to another goal visible from a distance.

It will not be my object to lead you along false paths over which the study of inflammation has travelled in the course of its development; I will confine myself mainly to sketching the line of thought which still urges itself upon us on a close examination of the anatomical data, and which science also in the course of its development has to some extent followed.

First, the question arises as to the old theory of inflammation.

Do the manifestations grouped about the vascular economy really designate the sum total of the processes of disease of an inflamed organ? We need only examine purely typical specimens of inflammatory processes seriously to doubt this assumption. In an acute catarrh of the mucous membrane we find the exudate by no means built up exclusively by transudation and emigration from the vascular system, but that the increased secretion of mucus by the mucosa, as well as the proliferation and desquamation of its epithelia, also plays an important *rôle*. The latter manifestations are, in fact, characteristic of catarrhal inflammation; degenerations, especially mucous degenerations of the desquamated epithelia and of the emigrating cells, are certain to occur. Hence, even in typical inflammations, such as acute catarrh of the mucous membrane, we must admit that the inflammatory vascular alteration constitutes but a part, even though an important one, of the entire condition of disease.

The diseases of entirely or partly non-vascular parts—cartilage, cornea, cardiac valves, endocardium—likewise point to a similar conclusion. These are organs in which the complete picture of the inflammatory vascular alteration is excluded, because its anatomical conditions are wanting, or appear only in a secondary manner by the immigration of leucocytes from neighboring tissues or by the subsequent vascularization of the originally non-vascular tissues; it is difficult, however, for us not to think that those changes which we observe in the first stages of keratitis, choroiditis, and endocarditis are the really important and essential ones for inflammation, which is, in fact, assumed in their very names.

The nature of these changes need only be indicated here. At first, and in many cases perhaps continuing so, they consist in a swelling and enlargement of the cells, which give rise to granular opacity and fatty degeneration, on the one hand, but, on the other, also to their division and multiplication and to the formation of amœboid derivatives of fixed cells, while the greater number of the migratory cells which appear are the leucocytes emigrating from the vicinity. In acute endocarditis we find from the beginning a cell-proliferation and a layer of granulation on the cardiac valves, which then vascularizes.

We are compelled to similar views by certain acute affections of so-called *parenchymatous organs,* as, for instance, the kidneys, liver,

heart, etc. Through the action of agents known as inflammatory irritants these organs suffer more or less extensive changes of the parenchyma,—that is, of the epithelia of the respective muscular fibres,—changes which consist essentially in granular opacity, fatty degeneration, and even death; in such cases the above-mentioned manifestations of inflammatory vascular alteration are either altogether absent or are so feebly expressed that they are completely overshadowed by the degenerative processes. Such affections, in which the degenerative factor so completely predominates, have been called *degenerative inflammations.* To these belong a large percentage of cases of acute nephritis, in which the most various degenerative processes take place in the cortex of the kidneys without necessarily incurring any marked changes in the circulatory apparatus, and in which the cortex of the kidneys is usually found to be anæmic, while the medullary substance, which is affected by the disease to a much smaller extent, shows a congestive hyperæmia. In the cortex the signs of inflammatory vascular alteration are often confined to slight small-celled infiltration of the interstitial tissue and of the glomeruli, coagulated serous discharges into the *tubuli uriniferi,* where they form a part of the hyaline cylinder, and perhaps also small hemorrhages. In the main, then, in a large number of cases at least, it is a condition of nutritive disturbances in specific organic elements, called by Virchow conditions of irritation with subsequent change into degeneration, but at present almost universally considered to be purely degenerative processes. In other cases an acute inflammatory process passes into a chronic condition, not without in certain respects changing its character. If we refer again in the first place to the catarrhal inflammations of mucous membrane, we notice that in its chronic forms there is a decrease in the intensity of those processes known as inflammatory alteration; on the other hand, in chronic hyperæmia there sets in more and more a prolonged dilatation, thickening, elongation, and hence spirality of the blood-vessels, especially of the veins; there may even be formation of new blood-vessels. Instead of the infiltration of the tissue with multinuclear or polymorphonuclear leucocytes supervening in acute inflammation, there appears more and more uninucleolar small round cells, the origin of which, whether they are from the blood or from the lymphatic system, or derivatives of proliferating connective-tissue cells, is still an unsettled question.

But other processes gradually assume a preponderance: proliferation and formation of new glands, thickening and compressing of the interstitial connective tissue,—in short, processes of an essentially tissue-forming, productive character. Hence the infiltrative condition passes more and more into that of hyperplasia of the tissue; but these essentially hyperplastic conditions are still designated as inflammation. Since, however, the proliferative processes here form the principal factor, we speak in such cases also of productive inflammation. In some cases such a state passes ultimately into purely atrophic conditions, in which the proliferation of connective tissue results in the formation of a hard, dry, cicatricial tissue, poor in blood-vessels, and, as for instance in mucous membrane, leads to its reduction, smoothing, and shrinking, with loss of the glandular apparatus.

With the recognition of a degenerative and a productive inflammation a radical departure was made from the original conception of inflammation. This conception has thus become wider, but also less definite. This, however, has brought us to a sloping road, since we are now logically compelled to consider as productive inflammation also proliferating processes in which the inflammatory disturbance is present from the start, but appears only in a very small degree. Such are the so-called chronic interstitial inflammations, to which belong interstitial hepatitis, nephritis, and others, as well as many productive inflammations of the serous membranes, where from the beginning there is scarcely any formation of exudates, but essentially proliferation in the connective-tissue framework of the organs. As to chronic interstitial inflammation, it remains to be said that the principal condition common to all the disorders classified under that head can arise in various ways. I shall mention here only two examples, the so-called fibrous myocarditis and the picture of disease called chronic endometritis.

The first name indicates nothing but that bands of connective tissue have been formed in the muscular system of the heart, that this has caused the destruction of muscle fibres, and that a cicatricial formation has set in. But in what ways can such a cicatrization arise? Anæmic necrosis, hemorrhages, softenings, true (acute or chronic) inflammations, thickening of the perivascular connective tissue, atrophy or tearing of muscle fibres by the over-exertion of the heart—all these may be causes of cicatricial formation. To be

sure, this is a case where the name given the above condition indicates only the final effect common to various processes.

Very similar probably are the circumstances in the case of chronic endometritis, the "infarct of the uterus," although our knowledge of this disease is much smaller. In many cases the process is connected with endometritis, an inflammation of the endometrium encroaching upon the muscular system of the uterus, which leads to exudative and infiltrative manifestations, with degeneration of muscle fibres, and finally to the formation of connective tissue. The principal anatomical change in metritis seems to consist in an increase of the interstitial connective tissue within the uterine wall, and, according to more recent investigation, it would appear that the relative preponderance of the interstitial tissue over the muscular system is the condition to which must be traced the symptoms of disease. But such a disproportion between the two forms of tissue can arise from various causes. Sometimes hyperplasia of the muscular system is the cause, as in the infantile uterus; in other cases the origin of the "atony" of the organ is perhaps traceable to an atrophy of the muscular system of the uterus; congestive as well as obstructive conditions in the uterus, disorders of the adnexa, and other disturbances can also stand in causal relation to so-called chronic metritis. At any rate, there is no uniform process in the beginning of the disease, but there is a final effect common to various processes.

When we recognize in these conditions the idea of a degenerative and an interstitial inflammation, we have dismissed the factor which at first appeared to be the most essential characteristic of inflammation,—*inflammatory alteration* in the blood-vessels; for in many of these forms this factor is altogether secondary. Moreover, with this the word inflammation loses its significance as conveying one definite idea, and becomes merely a collective name for a number of various processes, no longer indicating a peculiar quality common to all the conditions comprehended by it, and abstracted from them and from them only. The various partial manifestations in these processes are at the same time also partial manifestations of other processes,—of reparative and healing processes, of hyperplasia, even of tumors, as well as of regressive processes; since vascular alteration is no longer accounted a characteristic of the theory of inflammation, the common bond separating inflammation from the

above-mentioned processes ceases to exist. Another characteristic common to the now larger and more extended group, and including parenchymatous, exudative, and productive processes, became necessary, and this was found in the etiological element leading to inflammatory manifestations,—namely, *irritation.*

The assumption of a *primary irritation* of the tissue-cells is the foundation of Virchow's theory of inflammation. According to Virchow, the cells are excited to increased activity by the action of the inflammatory irritant, which manifests itself in a twofold form,—the nutritive and the formative; the cells are enlarged by an increased absorption of albuminous material, which in certain cases is transformed into fat and other degenerative products by a change in the functional activity of assimilation; finally, the cells may be completely destroyed (parenchymatous degeneration resulting in fatty degeneration and decay), or the cells are excited to division by irritation, giving rise to cells of the same kind or pus-corpuscles. The circulatory disturbance of inflammation Virchow considers a secondary process, the increased nutritive requirements of the irritated cells causing an increased supply of nutritive material,—that is, of blood.

Marchand recently pointed out that the theory of Virchow is not a material departure from the original theory of "irritation." Originally by irritation was meant the external cause which liberates the physiological function of an organ or of a tissue element, and the word is still used in physiology in this strict sense: the irritant affects the nerves, muscles, mucous membrane, and produces excitation of the nerves, muscular contraction, or secretion; throughout life we are subject to continual irritation. An inflammatory irritant, on the other hand, which also can only be physical or chemical in its nature, is one which leads to peculiar changes in the tissue elements and may excite the cells to quite heterogeneous action,—increased assimilation, division, formation of pus-corpuscles, etc.

Looking farther into pathology and the related sciences we meet with still greater differences. We see that the word "irritation," like the word "inflammation," is used in very different senses, and its meaning no longer corresponds with what Virchow understood by the term, although inflammation is still often defined as irritation.

In the first place, we know that chemical substances, traumatic action, dust, etc., act as inflammatory irritants, having the capacity to produce inflammatory conditions in previously normal organs. We must, however, not underestimate the significance which the specific constitution of the given tissue has for the manner in which the reaction takes place; not in every tissue does every irritant excite inflammatory conditions. Certain processes which must be regarded as healing processes are attributed to the action of irritation; thus, the infiltration with leucocytes and other migratory cells which gives rise to the resorption of fibrin coagula or extravasation of blood, or of dead, decaying tissue, is regarded as the effect of a chemical irritation produced by the dead masses. But even in the immigration of the large migratory cells, which soon appear in such preponderating numbers, we evidently have not merely an attraction but a new formation of those cells; hence there is cell division in the neighboring parts. This, as well as the proliferation of cells which bring about the restoration of the lost or dead parts of tissue, is regarded as the effect of the presence of the foreign or dead masses on the healthy neighboring parts; in the same manner the process of encapsulation of foreign bodies is regarded as the effect of irritation exerted by the latter. The entire healing process of the wound is by many regarded as an inflammatory process. In cases of extensive lesions which are perhaps also connected with contusion or necrosis of the edges it is possible, at any rate, to refer to chemical irritation, and this can be attributed to the blood or serum poured out into the gap of the wound or the dead portions of the edges. In the simplest case, however, the healing of simple, non-infected, incised wounds, with smooth edges, such action of irritation is scarcely present, there being only a restoration of the lost parts, as in the skin, for instance, of epithelium and connective tissue of the narrow wound gap. It is the same in cases where in any manner only individual cells or cell complexes have been destroyed; for example, individual muscular fibres through the known wax-like degeneration after typhoid fever, individual uriniferous tubules through the effect of toxic or infectious injuries, etc. In all these cases there is a pure regeneration for which we at present know no other adequate cause than the existence of a defect in the tissue.

In another sense, more closely related to that which it has in

physiology, is used the expression "functional irritation." It is known that such parts of tissue as functionally perform the greatest amount of work are also those which attract the most nutritive material; they grow and become stronger at the expense of other parts; thus, for example, there arises the functional structure of the bone, its architectonic form so adjusting itself that the trabeculæ of bone arrange themselves in the direction of the strongest incubus, the so-called lines of incubus, —that is, in the curves where the body-weight exerts the greatest pressure upon the bone. If under pathological conditions a change is brought about in the nature of the incubus, as, for example, in ankylosis, then with the change in the curves of pressure there is also a change in the arrangement of the trabeculæ of bone. A very apt illustration of functional effect is afforded by the development of the muscular economy of gymnasts; the cases of so-called compensatory or "labor" hypertrophy. To this group belong hypertrophy of the heart due to more work being demanded of it to overcome increased resistance in the circulatory stream beyond the place where the hypertrophy occurs; unilateral hypertrophy, which sets in after suspension of functional activity of one of the double organs, as, for example, in the remaining kidney after the surgical removal of its fellow; and the so-called "*regenerative hypertrophy*" as in the liver, which may be almost completely restored to its normal size even after complete destruction of four-fifths of the hepatic tissue. In these cases we may have to deal also with other etiological factors, such as functional irritation causing regenerative processes in the epidermis. In new-born animals where unilateral extirpation has been made of glands which begin their function in later life, such as the testicles and the mammary glands, hypertrophy of the remaining gland sets in when the time for the functionating of the gland arrives.

The above facts seem to prove that functional irritation cannot be considered an adequate cause either of compensatory-hypertrophic or of regenerative processes, and in some cases cannot even be admitted as a partial explanation; and, as the effect of the knife, *per se,* in making the wound cannot as an irritant excite regeneration, there remains as the true cause of the latter only the existence of an interrupted area or a defect.

In embryology also we find the conception of irritation to be a

very broad one. When a portion of the wall invaginates on the blastula and thus becomes the entoderm, we speak of the " action of irritation," which is perhaps caused by a peculiarity of the invaginating wall portion, as, for instance, greater osmotic pressure, and hence its swelling. When an external cause, a firm wall, for instance, gives rise not to an outer but to an inner folding, then the resisting wall acts as an irritation.

In short, the word irritation has a different meaning in the different special sciences. The physiologist understands it differently from the pathologist, and in embryology it is used in quite a different sense from that which the practitioner has in mind when he speaks of a " tissue in a state of irritation;" the latter conception is, on the whole, equivalent to Galen's symptom complex.

Irritation was spoken of above as a " liberating cause." Let us see what this means. When in any phenomenon, by no means necessarily *vital,* the cause is quantitatively altogether out of proportion to the effect produced, it is spoken of as " liberation." Thus, the discharge of a gun by a slight pressure of the finger is called liberation; in the same manner we speak of liberation when a single thrust causes the explosion of the chemical energies stored up in a vessel of powder, or when a stone supported on a tower is made to fall to the ground by being pushed over the edge. In all these cases the process causing the various effects, the applied force which liberates them, stands in no relation to the effect produced; the question is merely one of the passing into action of a latent force, this force being already prepared in the system in which the process takes place, and lying in readiness, as it were, so that only a slight impulse is necessary to set it free. Hence, although an external cause is the condition of every event, it represents but the last one of a series of conditions which must be fulfilled if an effect is to take place, but which takes place immediately on their fulfilment.

Strictly speaking, then, a liberating cause is not a cause by itself, but only a partial cause, one of the necessary conditions of a given phenomenon. This, in fact, is true of all causes; for in all the known phenomena the real cause does not consist in a simple process or condition, but in all the conditions necessary to produce a certain phenomenon. In language, however, and generally also in science, cause in the narrower sense designates that process which

has appeared last and which is immediately followed by the effect, and thus seems to us as the most important condition (partial cause), the one immediately giving rise to the event. Notwithstanding the fact of the universal use of the word cause even in its strictest sense for only a partial cause, and that the difference between cause and liberating cause is only a relative and quantitative one, we must, nevertheless, bear in mind that in the case of the latter the disproportion between the immediate cause and the energy liberated can be given.

The second point is that what is designated as liberation cause and, as I believe, not very happily, also understood by the word "irritation" is not infrequently but a negative element, as in many cases of regeneration, when the only possible liberating cause to be assumed is the existence of an interrupted area, or the lack of a normal neighborhood. If, on removing an extremity or the tail of a newt, the extremity or the tail grows again, the immediate, external cause of this is only the presence of a defect; if, further, after the disarticulation of the femur in the same animal, the bone does not regenerate, it proves that for bone regeneration it is also necessary that there be an interrupted area,—the wound area; the liberation, the "irritation," for the regeneration of the bone is thereby only more precisely defined, but the immediate external cause is still only negative; the sum of all the other positive conditions, the real cause, in other words, was in the tissue itself, and constitutes an unknown quantity, which we denominate as capacity for regeneration; liberation only sets this in action.

If, therefore, there are cases where only internal causes can be assumed to explain regeneration, it is highly probable that these same internal causes, the "formative impulse" (*sit venia verbo*), when free to act, is the true etiological factor also for the other regenerative and compensatory processes; which assumption does not negative or even reduce the importance of *functional* or other irritation as a liberating cause. Finally, it is a liberating cause of a negative character that is assumed also when, with Weigert and other investigators, we speak of a proliferation of tissue through atony, or the cessation of a state of equilibrium between the individual tissue elements. Strictly speaking, here also we have but defined more accurately one condition, and that too a negative one, under which proliferation takes place. There exists a similar rela-

tion here to that of the so-called disburdening hypertrophy, when, for example, an articular cartilage hypertrophies after the removal of its antagonist, because there is no longer any physiological resistance to growth on the opposite side. As opposed to the above, it must be stated that inflammatory irritation in Virchow's sense represents a positive value, and, at least in the ordinary sense of the word, can be taken as the *cause* of inflammation. It is because Virchow's theory has become partly antiquated, though not entirely superseded, by reason of the new processes which are now classified under irritation, that I have here again presented the various facts of the case.

Subsequently, however, that conception was modified, and by many altogether abandoned; it was modified in so far as the degenerative disturbance of nutrition of the cells in inflammation is now no longer looked upon as a change of an active state of irritation, but as in itself a regressive manifestation. The existence of a formative irritation was especially combated by Weigert, who says that with its recognition an effect is ascribed to some chemical or physical agent upon the cells such as the sperm exerts upon the egg-cell.

As a consequence of this conception, all the cell proliferations and tissue formations in inflammation must no longer be regarded as phenomena of a regenerative character, or attempts at reparation and regeneration brought about by inflammation, but as taking place *after* the tissue lesion and even *despite* inflammation. In reality, therefore, their cause would not be a positive irritation, but, as we have already explained, a negative element. It is perhaps, however, not necessary to go so far as to deny the formative irritation of the tissue in inflammation. Marchand upholds the theory of the positive character of irritation; but it appears to me that even with this there is connected a certain change of the original view of inflammatory irritation. Two instances will suffice to prove this. Marchand shows, in the first place, that the cellular elements can be excited by different external causes; aside from the liberation of the function by specifically functioning, highly differentiated cells, the *general functions* can be liberated from the nucleus and protoplasm and can manifest themselves in a threefold relation: in *nutrition* (assimilation), *motion* (contraction, absorption of formed elements, locomotion), and multiplication. The exciting causes, the irritants,

are *physical* (mechanical, thermic, etc.), or *chemical,* while nervous influences can enter only in the more developed, highly differentiated parts which are connected with the nerve endings. Examples of the physical action of irritation are cases in which increased pressure gives rise to increase of cells; such is the thickening of the epidermis through external pressure and of the vascular wall by heightening the blood-pressure, in which cases there may also be an increased action of functional excitation.

In young epithelial cells the tendency to spread out on free surfaces depends upon a *tactile* irritation, just as migratory cells oppose themselves to fibres, fibrins, foreign bodies, etc. The importance of temperature for the growth of the organ is especially manifested in the experiments upon rabbits. The ear of a young rabbit which is kept in an artificially higher temperature grows more rapidly than the other; in the mature rabbit also there is an increased growth of the epithelia of the epidermis and sebaceous glands, which shows itself also in the presence of more numerous karyokinetic figures in these parts, while connective tissue and cartilage remain unaffected. Nevertheless, the effect of higher temperature is only partly a direct one; in part it is to be traced to an increased supply of blood. Chemical irritants can produce changes of place of the migratory cells and changes in form of originally fixed tissue-cells, with greater accumulation of protoplasm at definite parts of the cell exposed to the irritation; according to Marchand, there is connected with this an increased growth, which, according to experience, does not lead to an unlimited enlargement of the cells, but after a certain limit has been reached, to their division or proliferation.

There must further be mentioned the *chemotactic* action of many substances upon tissue-cells. This is not the same that has been long known as exerting an attraction on the leucocytes; especially *dying leucocytes* and *organic substances* in general, as well as *fibrin,* that cause chemical irritation in the near-by tissue-cells, and thus give rise to their proliferation and to the formation of amœboid derivatives. Finally, Marchand deduces as a very remarkable but scarcely controvertible fact the mutual attraction of the same tissues, as, for example, of nerve-fibres or epithelia.

If, however, we ask how far, in the examples adduced above, irritation may be regarded as the real cause of the formative activity

of the tissue, the answer, I believe, will be that it is so only in a very limited even if positive sense. When by the application of higher temperature the growth of a young rabbit's ear is accelerated, it is in reality only a *furtherance* of a process which is already in action, and not a real liberation or a *new* process. So likewise is it but a promotion of growth in those experiments on mature rabbits in which by the application of temperature to one ear a cellular increase is excited in the epithelium of the skin, in the sebaceous gland, the hair, etc. (but not in cartilage), for we know that these tissues show mitoses also under physiological relations, even in the grown organism, which points to a continued process of physiological regeneration of the impaired parts; but by the increase of the blood-supply the growth can partly be promoted, just as by hyperæmia and consequent increased supply of nutrition the secretion of a gland can be strengthened, and it is not necessary to assume the presence of the irritating substances as the sole cause for a specific secretion; they only release the secretion on the cells adapted and arranged for that purpose.

Similarly the capacity for the complicated processes of *cell division* must be contained in the cells, if this be the result of a chemical or physical irritation; for otherwise the excitation to division would really be equal to the irritation of impregnation. There must, therefore, be a middle process between the increased assimilation of the cell excited by external irritation and its consequent increase, on the one hand, and the division connected therewith, on the other; and this middle process must be a cause within the cell itself. In other words, even in cases where a positive irritation really gives the impetus to the activity of cells, it acts only as a liberating cause.

From what has been said it may be concluded that very positive irritants can be recognized as causal elements in the formative processes; but it must be remembered that they represent only liberating causes the effect of which can be brought about in other cases by negative elements (decay of parts, etc.); nor will there always be but one liberating cause; we must rather assume the combined action of several causal elements; least of all ought this to be assumed in many pictures of disease known to us that depend essentially upon formative irritation. A brief consideration of some processes generally denominated as production or interstitial

inflammation, and of a few others not anatomically distinguishable from them, will convince us of this. As examples of such diseases we may cite interstitial nephritis, interstitial hepatitis, and chronic, productive inflammations of serous membranes. The first two, which take place in so-called parenchymatous organs, show, on the one hand, a degenerative process in the specific tissue parts, in the parenchyma of the liver and the secreting parenchyma of the kidneys, partly also manifestations of proliferation of the secretory cells, and, on the other hand, a proliferation of the interstitial connective tissue. Those processes in the vascular apparatus which are so prominent in acute inflammation are very slight in these forms, and would in themselves hardly be thought of as inflammatory in their origin.

Formerly these were classified under *primary interstitial inflammations,* but now it appears that not infrequently quite similar forms are associated with more or less acute diseases, which sometimes set in even with manifestations of inflammatory vascular alteration; that, further, the very same anatomical pictures can arise if in some manner—as, for example, through the obstruction of the blood-supplying vessels—the nutrition of the parenchyma has been impaired.

As to the relation in which the individual component parts of these anatomical pictures stand to each other we may suggest various possibilities.

1. As the first group of cases belonging to this category we may name those in which a *reparatory proliferation* in the sense above spoken of is present in the interstitial tissue, and hence the directly liberating element has an active character. To this belong the scar formations in the kidney after anæmic necrosis of its epithelia, as well as in infarcts; a typical example is further offered by the proliferation of glia which arises in the central nervous system after the destruction of nerve-fibres, and leads to the formation of true glia scars. This process is similar to the spacial reparation of regeneration, the difference being that the specific tissue has not sufficient capacity for proliferation to cover a defect completely and so there arises in its stead supporting tissue. It should be noted, however, that this proliferation is not designated "inflammation" by the practitioner: a scar which marks the place of a healed infarct in the kidney is no more called nephritis by the clinician than the

glia proliferation which attaches itself to secondary degeneration of the spinal cord is conceived of by him as myelitis.

2. Such reparatory interstitial proliferation, however, certainly plays a great part in many cases of so-called chronic inflammation, in a large percentage of contracted kidneys, in cases of atrophy of the liver and other organs. One need but recall the so-called secondary contracted kidney, in which through toxic or other causes the degeneration of the parenchyma takes place and interstitial proliferation only gradually follows here and there; some authors even go so far as to regard interstitial proliferation in all cases as secondary, explaining the progressive interstitial process by continual destruction of new parenchyma, which in turn gives rise to continuous reparatory proliferation; in reality, therefore, this would constitute a continuous cicatricial healing of patches of degeneration continually arising. A few other contractions of the kidneys or the liver are no doubt to be explained in the same or in a similar manner. Thus, for example, a contracted kidney may be caused by venous obstruction in such a way that through the dilatation of the capillaries and the increased pressure of the veins a compression of the tubuli uriniferi takes place, giving rise to atrophy of the secreting epithelia, which in turn leads to interstitial proliferation.

3. This, however, does not exhaust the field of interstitial inflammation; for the present, at least, I do not believe we are in any way justified in excluding the possibility of a direct external action, of a toxic character, for example, for the excitation of the proliferation of interstitial tissue,—that is, for the bringing about of a direct action of irritation in the sense of liberation. But how much in a particular case is to be attributed to such direct action of irritation and how much is to be accounted as reparatory proliferation is at present entirely beyond our knowledge.

We thus see, from a consideration of the forms described above, that different exciting causes act as irritants, the *rôle* of which in particular instances we have yet no means to ascertain, that negative and positive irritants often act together, and that the latter have nothing specific about them. An inflammatory irritation, as such, can, therefore, as yet not be distinguished from the various other forms of irritation, and thus even by the assumption of an irritation we have lost the criterion we hoped to be able to find as characteristic of the process of inflammation. At the same time there is no

possibility of distinguishing inflammation from other processes, at least from the causal stand-point; for in the last analysis the action of irritation is common to all biological phenomena,—to regressive metamorphosis as well as to formative manifestations; even the picture presented by a fresh fixed cell under the microscope may be considered as a product of the action of irritation,—that is, the action of the last irritant which the fixation liquid has exerted on the cell at its death. Above all, it is quite impossible, from this point of view, to separate inflammation from other progressive processes of a regenerative, reparatory, and hyperplastic character.

We have frequently spoken of internal causes, indicating thereby characteristics and capacities not carried from the outside into the cells but inherent in them. Let us see whether in this relation there is in inflammation a change of the biological process, perhaps one even which is characteristic of it. We will test this question in a form of disease which in all respects is a typical inflammation and not seldom passes from acute stages into a picture of chronic inflammation: catarrhal inflammation of the mucous membrane.

What takes place in the acute form in the vascular apparatus does not consist, as we have already asserted, of new manifestations foreign to the tissue, but such processes as are already given under physiological conditions, but which in inflammation appear in increased measure and in a modified form, as hyperæmia, transudation, and emigration. If we assume with Heidenhain that the passage of serum through the vessel wall does not depend merely on infiltration, but largely on the secretory activity of the endothelium of the capillaries, which can be increased by chemical agents ("Lymphagoga"), then, as regards inflammatory transudation, we could speak of a state of irritation of the circulatory apparatus with production of an abnormal, richly albuminous secretion. That this does not explain the whole of the inflammatory circulatory disturbance is self-evident; even emigration is principally to be attributed to other so-called chemotactic influences which work from the outside directly upon the elements of the blood capable of migration.

On the other hand, the rest of the tissue of the inflammatory mucous membrane shows very plainly the state of irritation or state of the diseased excitation of the tissue. The greatly increased mucous secretion, the proliferation and desquamation of the epithelium which imparts to the inflammatory exudate not only the

character of the transudate but also partly that of a tissue product, are signs of the increased reaction. In the desquamation and mucous degeneration of many cells there is further evinced a tendency of the irritated tissue to retrogressive metamorphosis, a change from the state of irritation into degeneration. There is present, therefore, that which might be called a heightened feverish activity of the inflammatory mucous membrane; and the individual component parts of normal life show themselves in various and disproportionate measure. Klebs rightly designates this condition not only as a simple, heightened reaction of the tissue, but as one of a diseased modification. There is, however, a second very important point to be considered here. The ready irritability of the catarrhal mucous membrane, as of inflammatory tissue in general, is known; in catarrh of the nose the smoke of a cigar is sufficient to produce a sudden increase of the catarrhal manifestations, increase of the swelling, increase of mucous secretion, small hemorrhages and even heightening of the general affection of the body. Hence, not only is the mucosa in a state of heightened reaction, but its capacity for reaction against new irritants is increased. We must also note that the duration of tissue reaction does not by any means entirely coincide with the presence of the exciting cause. When once an irritation has set in the reaction continues even after the action of the primary cause has ceased.

When a catarrh of the mucous membrane becomes chronic, the manifestations in the circulatory apparatus as well as in the tissue itself gradually change. In general there is a reduction of the alarming symptoms in the circulatory apparatus; the congestion is slight, and there appears often an infection of the venous vessels, only more spotted, instead of the strong arterial hyperæmia. The transudation and secretion also decrease, at times suffering exacerbations; the microscopic examination reveals instead of the former animated emigration of the leucocytes, a preponderance of infiltrates of uninuclear, small, round cells, which must not all be positively traced to the blood, for they are to be considered as largely if not preponderatingly derivatives of lymphoidal elements and even of fixed connective-tissue cells. The composition of the inflammatory secretion in chronic catarrh is also quite different from that in the acute stage.

Further, the changes in chronic catarrh of the tissue of the

mucous membrane are essentially different from those of the acute stage, of which there is generally a very great variety. In general it may be said that near the aforementioned round infiltration, proliferations of the glands and of the interstitial tissue arise which gradually lead to a transformation of the structure of the mucosa; I mention only chronic endometritis with its various forms (glandular, hypertrophic, fungous, polypoid formation, etc.). The final result of many chronic catarrhs is represented by an atrophy of the mucous membrane with loss of the glandular parenchyma and cicatricial transformation of the hyperplastic interstitial tissue, a condition by which the specific functions of the mucous membrane are destroyed or, at least, are reduced in a corresponding degree. The reactionary state passes into a condition of atrophy.

We have observed above that the inflamed mucous membrane is not only in a state of increased reaction but also of increased sensitiveness to slight external irritants; we know that such an increased capacity for reaction continues for some time after an acute catarrh, and that often, through acute catarrh, produced by artificial means, may lead even to a certain disposition of the tissue to renewed disease. If within short intervals new catarrhs repeatedly arise so that the irritability of the tissue is almost constantly increased then there is produced a chronic condition which really consists merely of recurrences of acute inflammations.

In the real chronic catarrhs of the mucous membrane in which the permanent manifestations have set in on a higher level than the normal, the increased sensitiveness and the greater irritability of the tissue is easily determined, and shows itself in the violent flaring up of the florid inflammatory manifestations on the action of even very slight external irritation, and by the repeated acute exacerbations of the process, which takes place in a more and more incomplete form after each new attack. So, likewise, there remains a heightened disposition to influences which act in a different manner, for example, to colds. We know further that in cases where chronic catarrhs are finally cured, such a state of irritation continues for a long time, if not permanently; it is true, that in a large percentage of these cases there arise secondary affections, which have developed upon the area predisposed by the chronic inflammation and create a vicious circle. In many cases, as for instance, in many gonorrhœal inflammations, it is the original

infectional excitants which cannot be overcome by the passage from the acute inflammation into the chronic stage. In a chronic gonorrhœa a state of irritation persists even after the gonococci have disappeared or are present only in very small numbers; but there has remained the capacity of the inflamed mucous membrane to react again with acute catarrhal manifestations at the slightest injury, the reaction having become somewhat modified, according to the changed condition of the mucous membrane.

It can be stated, therefore, that in chronic catarrhs of the mucous membrane, as well as in acute conditions, a change in the capacity of the tissue for reaction takes place. The irritation swelling, that is, the mildest irritation which can be produced by any reaction, has decreased, and another lower resistance of the tissue to external influences has developed. Such a heightened irritability, however, and consequently capacity for reaction or heightened vulnerability, need not be a consequence of a past acute inflammation. Often enough chronic catarrh develops without a previous acute stage, and apparently under the influence of an inner disposition which affords a favorable field for the inflammatory excitants, while they would be powerless against the normal tissue. It is true these relations have been largely overlooked because disposition or tendency does not grow on culture media and cannot be stained with any color or shown under a microscope. Disposition is present as an unknown factor; and it seemed for a long time as if this element would vanish in the flood-tide of bacteriological discoveries.

It is not so long since scientists thought they could explain scrofula with the phrase that it was "the tuberculosis of childhood;" and yet it was recently demonstrated that in a large percentage of scrofular cases there is at least at the beginning no evidence of the presence either of tubercle bacilli, or of the typical anatomical changes of tuberculosis, and that in a great number of cases there is present an infection with the usual pus excitants, which settle upon a peculiarly disposed area and there give rise to various changes. The various affections of the epidermis and the mucous membrane that appear in scrofula are by no means all of tuberculous origin, measured by anatomical or etiological standards; whereas, there can be shown frequently the presence of other, especially pyogenic, micro-organisms in the affected regions. Such conditions favor a return to the older view of scrofula, according to

which these affections correspond to real tuberculosis only in part of the cases, but otherwise are to be regarded as congenital, constitutional anomalies, with heightened disposition to various infections, especially to tuberculosis; such a disposition is peculiar to childhood in general, but particularly to scrofulous individuals. In many cases the skin and mucous membrane diseases constitute the passage of entrance for the tubercle bacilli, and then develop like the glandular affections into real tuberculosis, which is thereby, as it were, ingrafted upon scrofula. The characteristic vulnerability of the scrofulous organism is, perhaps, to be attributed to a peculiar anatomical structure of the parts chiefly affected, to the looseness of the submucous tissue, to lymphstasis, and to greater capacity for the absorption of various bacterial poisons.

It appears that very important contributions to this subject have been made by more recent investigations on the pathology of the female genital apparatus, in those forms of chronic endometritis which accompany other diseases of the uterus itself or of other organs which are functionally related to it. I would mention in this connection the chronic endometritis which occurs in myomata and fibromata of the uterus, in carcinoma of the portio vaginalis, in stenosis of the orifice, and further in the displacement of the uterus. Likewise diseases of the ovaries and of the Fallopian tubes, as well as diseases of the adnexa in general, are accompanied by chronic endometritis. The connection of changes of the uterus with the primary disease is still to a large extent not clear, yet we must ascribe a significance to the circulatory and nervous influences, which play such a great *rôle* in the relation of the above-mentioned organs to the uterus. This is especially indicated by the relations between the functional processes in the ovary and the circulatory processes in the uterus and its mucous membrane. General venous obstruction as well as congestions can play a similar *rôle* to the uterus for the formation of endometritis to what obstruction (stasis) does for the recovery from gastro-intestinal catarrh and on other mucous membranes in heart diseases. Disturbance in the retrogressive metamorphosis of the decidua after normally or prematurely ending pregnancy, in which not seldom lobules of deciduous tissue remain, can also lead to endometritis like a tumor of the uterus. Our assumption of the non-infectious nature of such forms of endometritis is based essentially upon clinical observations, which

naturally cannot be as exact as the results to be obtained through combined clinical, anatomical, and bacteriological investigations; nevertheless, no practitioner should dismiss it offhand. Even if we assume that many of these forms depend on an infection—we may admit this for all of them without change of our view—all the clinical conditions show that this infection plays but the secondary *rôle* of an occasional cause, while the disposition located in the disease of the genital apparatus must have been the original determining factor.

The most significant result of these observations is that at least in chronic catarrhs developing from acute ones there is manifested a progressive preponderance of the intrinsic causes over the external liberating irritations. The increased proliferating capacity (which term may be adopted as the most convenient expression for the inner causes) is found, nevertheless, not only in inflammation but passes from this in increasing measure to other processes; it appears in a heightened degree in many forms of hyperplasia, even in such forms for which we know no external cause, but must assume a predisposition, congenital or acquired, as in many cases of elephantiasis, giant growth, adiposity, and of tendency to acromegaly. To a still greater extent this increased capacity for proliferation manifests itself in malignant tumors, which not infrequently develop from inflammatory processes, as, for example, carcinoma following lupus sometimes arises from ulcers of the gastro-intestinal canal and of the leg, etc. The point at which a non-typical epithelial proliferation, from which in such cases cancer arises, passes into a malignant or carcinomatous proliferation cannot be precisely determined, and it is only possible to recognize that the change had commenced when the proliferation penetrates into the deeper layers of tissue. This is the position, at least, if we assume as the most significant factor for the development of malignant swellings an inner change in the proliferating capacity of the cells,—an anaplasia as Hansemann calls it, or *cataplasia* according to Beneke's designation.

The case would not be very different if Ribbert's theory held true: that in carcinoma the displacement of cells by other cells, of epithelia by connective tissue, and their transferrence to other nutritive relations could, under certain conditions, establish a pronounced capacity for proliferation. In that case also it would be

merely a demonstration of inherent capacities of the cells; should, however, the search for infectional excitants in malignant tumors, which has been pursued for decades with the greatest zeal but thus far without result, be finally crowned with success and reveal some exciting parasite in carcinoma and sarcoma, then the theory of the inner causes of malignant tumors would, for a time, at least, drop out of discussion, just as the theory of disposition to tuberculosis was eliminated by bacteriologists, according to strict observation, after the discovery of the tubercle bacillus—perhaps only to assume later a still greater significance than has been ascribed to it before. It would lead too far and not be in place here to discuss the grounds which cause many pathologists at present to think it very improbable that a parasitic excitant of carcinoma or sarcoma will ever be discovered, because they do not consider these diseases to be of a parasitic nature. But it is possible that, contrary to all expectations, such a discovery may one day be made; even in such a case, however, it still remains to be seen what else such an infectional excitant could do but give definite direction to the inherent tendency of the cells arising from inner causes.

Recognizing the impossibility of distinguishing between the diseases designated as inflammatory and others, Thoma proposed to abandon the conception of "inflammation" entirely; all those names which by their ending *itis* indicated that the diseases designated by them were to be considered as inflammatory he would divest of this signification, and let the term mean simply disease—a proposition which when closely considered is not so greatly at variance with what is the actual and usual conception of inflammation in a broad sense. Thoma has also shown by the example of kidney diseases that it is quite possible to retain the old terminology while dismissing the conception of inflammation. The objection to this was that the conception of inflammation has proved of great heuristic value, and that in the endeavor to explain the theory of inflammation a number of works had sprung into existence which have brought many positive and valuable results. Admitting this, it is, nevertheless, difficult to understand why a conception which is not well defined and which essentially is based on false reasoning should be preserved simply because the questions raised led to works which would sooner or later have been accomplished independently of this conception.

The chief objection that can be raised against Thoma's suggestion is that the clinician could not get along without the conception of inflammation. It seems to me, however, that even from the clinical view the ambiguous application of the word "inflammation" has caused no little harm. Just as the application of the word in the clinic indicates more than merely the external, visible changes, so in internal medicine, it leads to still greater ambiguity than in pathological anatomy. Witness, for example, what the conception of myelitis, "inflammation of the spinal cord," has become in the hands of the clinicians: a symptom complex, which cannot be disposed of in any other way, that is all. Whether in myelitis the marrow is inflamed or not is usually no longer discussed; the clinicians and neurologists designate what they rightly call anæmic or hemorrhagic softening in the brain simply myelitis if it is in the spinal cord, because their point of view does not afford them any means whereby to differentiate these processes from myelitis, and the subsequent pathological-anatomical examination also leaves them greatly in the dark; if an arteriosclerotic contracted kidney or a vascular contracted kidney caused by lead poisoning leads to atrophy of numerous glomeruli and thus gives rise to manifestations of ordinary, genuine contracted kidney the clinician would unhesitatingly call this a case of nephritis. Here, therefore, still more than in pathological anatomy, the door is left wide open for confusion.

In spite of this, the clinicians, especially the representatives of surgery and of allied branches, will not allow themselves to be deprived of the use of the word inflammation, and I believe with good reason. It will, however, be essentially a matter of those cases in which the inflammation as such can be diagnosed by clinical methods, which amounts to the application of the Galenic symptom complex; when the surgeon says that the lips of a wound are inflamed he does not designate the process of the healing of the wound, but only an accidental manifestation caused by a specially strong development of the individual, partial manifestations of the same; he does precisely what I have suggested above, that is, designates by the word inflammation not a complete anatomical process, but only an attribute of it, and no more.

We have attempted to subject the symptom picture of inflammation to an anatomical analysis, and on the basis of such an analysis

to define a theory of inflammation. It appears that the anatomical manifestations which are the basis of this symptom complex do not correspond to the whole of the disease but only to a part, varying in extent in different cases. Our deductions lead us to agree with Weigert that it is quite impossible to distinguish inflammation from other subjects of pathology; we are compelled to admit that no element common to all the so-called forms of inflammation can be found which, as "irritation," cannot be found also in other pathological processes. This is nothing new. The best confirmation of such a deduction is afforded us by the words of Virchow at the Moscow International Congress: "Inflammation as it can be at present defined is no uniform process having constant characteristics." But we can never hope to be able to find a sign characteristic of all forms of inflammation and of inflammation only; we must rather conclude that the extended conception of inflammation introduced by Virchow was a reasonable postulate in the light of pathological knowledge then existing, but later research does not warrant its acceptance. If this is true then there is nothing left but to follow again the road, which had been traversed but had been deemed impracticable, back to the point of departure from which we have begun our analysis—the symptom complex of inflammation.

What consequences would it have for our view if we recognize vascular alteration alone as the criterion for designating a change as "inflammatory"? The word inflammation will then cease to be the name of a species; that is, it will cease to designate one of the principal divisions of anatomical processes into which we divide the pathological phenomena when we say that the subject can be classified into the chapters of "Disturbances of Circulation," "Regressive Processes," "Inflammation," "Hyperplasia," "Infectious Granuloma," "Tumors," etc. There remain only complex manifestations which group themselves about the vascular apparatus; in the complete picture of a disease, as catarrh or nephritis, they constitute only a partial manifestation, which, indeed, lends a definite character to the entire process, but is not comprehensive, being an attribute which belongs to certain diseases in a more or less complete manner. It is like classifying all rocks which contain a certain mineral in one group according to this single characteristic, without regard to their composition otherwise—according, for instance, as they contain lime or silicic acid.

Something very similar to this is done when we designate as inflammatory all those processes which have the peculiarities of so-called vascular alteration in common; we select from among the pathological processes one that is common to every member of the group, and disregard other distinctive characteristics. To many such a system will be looked upon as a step backward; and in a certain sense it does look like the surrendering of a position which we perhaps thought that we had already attained. It can be shown that many other groups of pathological processes are not species or classes of a natural system, but derive a name from a common element, recognizable only from a certain, definite point of view. Consider, for instance, the processes which may be grouped under the name of reparative processes. When parenchyma is destroyed in a certain organ, and its regenerative capacity is not sufficient to restore the loss of a specific tissue, then connective tissue takes its place; in this manner wounds or defects in the kidneys, the liver, or muscles heal by the formation of connective-tissue scars. Again, in the central nervous system neuroglia tissue occurs, which may also be looked upon as a supporting tissue. In all cases, therefore, a special substitution of the lost tissue takes place at least to such an extent that none of the gaps or open defects limited by the specific parenchyma remains. The whole may be looked upon as a consummation on the spot of the process of regeneration, and like it is strictly to be classified as a healing process. In many cases the healing is further complicated by the fact that old necrosed tissue or decayed masses of tissue, or blood discharges, or fibrin, or foreign substances are absorbed, and are then substituted by scar tissue,—processes which have retained the name resorption and organization; to these belong the healing of anæmic or hemorrhagic infarcts, of foci of softening and blood centres, the organization of fibrinous exudates and of thrombi; finally, these processes are closely akin to the enclosing of foreign bodies by healing, resulting partly in the penetration of the object with living cells, partly in resorption of the foreign matter, and partly in its encapsulation by the surrounding tissues.

In all these processes we see either a restoration of the normal quantity of tissue, even though often incomplete, or a condition established which in the long run can apparently not be supported by the organism (presence of necrosed masses, foreign bodies, etc.)

by another which is irrelevant for the organism. All these healing processes are especially characterized by the fact that they are not progressive, but remain at a stand-still as soon as a condition of possible permanence is reached. By maintaining the same point of view this group can be considerably enlarged so as to include a part of the hypertrophic processes. We often find processes which can be designated as "regenerative hypertrophy." If on the removal of four-fifths of the liver of a rabbit the remaining part of the organ enlarges by hypertrophy of its acini to almost its original size and, moreover, maintains its normal function, then there can be no doubt that regeneration is substituted by hypertrophy. The case is perhaps the same in cirrhosis of the liver where single hyperplastic acini develop between the atrophic lobes. When by increase of the blood circulation the heart hypertrophies and thus brings about a compensation for a valvular defect, or when on the extirpation of one of a pair of glands the other becomes larger and performs the whole function of the two, then these functional hypertrophies are certainly useful processes for the organism. The cause of such processes can be partly traced to a functional irritation, but as already stated in certain cases it has thus far been altogether impossible to find an external cause; as, for example, in those glands which begin to function only at a later period in life, as the mammary glands in young, newly-born animals, etc. These may be called processes of adaptation. In lower animals and plants such adaptations are present in still greater number and variety; I only mention heteromorphoses, those cases in which the organism in consequence of encroachment from without forms organs in positions where under normal conditions they do not belong, or in which lost parts are substituted by others differing from the first in form and function.

I may as well admit at once that the view-point under which the above-mentioned processes have been summarized is a teleological one, and that I have thus ventured upon somewhat dangerous ground which is at present very much disputed. I shall confine myself to the barest necessary limits in an attempt to justify this position. No one, of course, will suspect me of using the word teleology in its old sense, in the sense, namely, that there are in nature other causes beside the ordinary causes, "final causes," "ends." This view has been generally abandoned and with it the

impossibility is inferred of explaining the occurrence of a process by its "purposefulness" (adaptability) in so far as by an explanation we usually understand the pointing out of causes; we merely estimate in a purely descriptive form the single processes of a series of processes following one another, in their significance for the occurrence of a final effect as observed experimentally, or we determine the part played for the common final effect by each of the processes in their simultaneous collective action, also from experimental observation. The question of the cause of the individual processes, the real explanation of their presence is not touched. We have to deal merely with a mode of observation, which we apply every day in biology, but which is not less essential to us than the one which seeks to discover causes.

Hence, although this method of observation is distinct from the demonstration of causes it gives us an insight into the connection of things which is not afforded by causal investigation. When we consider the significance of the valves of the heart and of the veins for the normal circulation of the blood, and recognize that in general it is only by these arrangements that the process of circulation is made possible as it actually occurs, then we have certainly gained an insight into the mechanism of circulation, quite independent of the question as to how these adjustments have been brought about. We have thereby obtained an understanding of these processes somewhat similar to the manner in which we understand a machine when we have comprehended the collective working of its individual parts. On the whole, the knowledge which physiology gives us of the life of organisms is based in great part on such observation. Causally it is thus far quite impossible to explain why the digestive juices which the various glands of the gastro-intestinal canal and their appendages secrete are complements of each other, why it is that each organ takes from the blood just the substance which it requires for its upbuilding, and that the different organic systems of the organism act together in a coördinate manner. We cannot assert that the knowledge of these interrelations, defective as they may be, are valueless, and that they do not lead to a closer understanding of the adjustments of the organism.

In quite a similar way we can view the observation of the processes referred to above as healing processes. If in a case of insufficiency of the valves of the heart we see that, despite the increased

resistance, the circulation remains normal or nearly normal on account of increased work of the heart, and that the heart through hypertrophy of its muscles is capable of performing this increased amount of work permanently; when we see that in a contracted kidney in spite of the extreme atrophy of the kidney tissue the quantity of urine does not decrease but can even be increased, and further determine that such an increase is caused by the increase of the blood-pressure, which in its turn is brought about by increased work and hypertrophy of the heart, then we have doubtless acquired an insight into the combined action of the various factors, the significance of which we should otherwise not have understood; in a certain sense the fact is explained to us that despite the greater difficulty of circulation into the kidneys a normal quantity of urine can still be secreted. Without, therefore, in any way recognizing the teleological doctrine of the finality of the processes in question, we can say that these various processes have a definite significance as parts of the whole, and that the final results would not be attained if one of these composite processes should be lacking. It would, perhaps, be best to apply the word used in physiology in an adapted sense and say that each of the processes fulfils a certain function. The increase of the blood-pressure in chronic disease of the kidneys, permanently maintained by hypertrophy of the heart, fulfils the function of being an essential, determining element for the quantity of urine secreted; the hypertrophy of the left ventricle in stenosis of the aorta fulfils the function of allowing the evacuation of an approximately normal quantity of blood from the ventricle in spite of the heightened resistance; the organization of a fibrinous exudate fulfils the function of gradually removing the same and of substituting for it non-irritating cicatricial tissue.

As a mode of observation, then, the teleological stand-point, at least in the modern sense, is thoroughly justified, but we must guard against confounding the causal with the teleological mode of observation; that is, against considering any process as the cause of that process, because from empirical observation it has been shown to produce a certain effect essential or useful to an organism (has been shown to "tend to or aim at a certain end"). This is just the mistake which the old teleology has made.

This point of view is so much the more necessary in pathology as causal research often reveals to us but the liberating causes, and

leaves us quite in the dark as to the inner causes and as to the question why a certain reaction takes place in a definite manner. It offers no clue whatever to the question why, after the development of a defect or of some other disturbance of a part the young tissue springing from the edges of the wound not only proliferates, but again differentiates in the precise and well-defined manner of a complicated structure; why, for example, on the stump of a newt's tail that has been cut off not only a formless mass of young cells grow up again, but bones, nerves, muscles, etc.; why regenerated, peripheral nerve stumps again establish connections with terminal organs, etc. This point of view is, on the whole, very remote from the assumption of inflammatory irritation, but it represents a new element which is of primary significance for the processes of reparation and regulation.

Nevertheless, in a certain sense the teleological mode of observation is useful as a help in the endeavor to obtain a knowledge of causes, in so far as it may be regarded as a preliminary step, bringing to a point the connections of the different members of a composite process, which we are so far unable to understand; as in the tracing of a road, single intermediate stations are marked out first, and the close connection between them, the real execution of the road is left to the subsequent detailed working out of it. The teleological view, then, gives us, as it were, a clue which, indeed, does not discover to us the missing links of the biological data complex, but yet determines for the present the gross interrelation. Against teleology as a mere mode of observation there is, so far as I know, no objection at present on any side.

If we now return to our subject we can assert that among the processes which we have classified above as regeneration there are a number which doubtless bear the signs of inflammation: those of resorption and organization, and of healing over and around foreign bodies, processes in which the manifestations of the inflammatory vascular alteration are given with those of the formation of new tissue. If we take as a basis the single anatomical processes it is quite impossible to separate the reparatory processes from the inflammatory ones; on the other hand, the former stand in close relation to regeneration and regenerative hyperplasia. We come now to a question, which in my opinion has been discussed to little purpose—whether inflammation is a "purpurtive" process at least in

its beginning. If we avoid the word purpurtive and ask only whether the significance of inflammatory processes, their "function" in the above sense, is such that it can be regarded as regulative, because it is useful to the organism, then we must answer this question decidedly in the affirmative for a large number of cases; but we must as often admit a change into a permanent injurious action. I believe the question is put the wrong way: it resolves itself into a very simple statement when we say that certain processes which, in reference to their empirical course and final effect (provided that there is no further disturbance), can be regarded as processes of a regulative character, show on the other hand also an inflammatory character. The latter designation is taken from a different stand-point, from the kind of the anatomical processes. A distinction without difficulty is quite impossible, because in both series we do not deal with parallel processes but with the observation of partly different, partly the same processes from different stand-points.

We must further state the important fact that processes which at first had a distinct regulative character lose this character under certain circumstances; that is to say, they lose the character of a healing process and acquire an independent significance. Under such circumstances they can no longer be regarded from the regulative point of view; thus it happens that a fibrinous pericarditis, pleuritis, peritonitis, perimetritis, etc., does not conclude with a resorption of the exudate, but that the granulative proliferation continues for a length of time even after the substitution of the previously secreted fibrin, or that even a new exudation follows. If we imagine the whole process of exudation and of healing continually repeated then we have a chronic interstitial inflammation. But there often appear on the serous membranes processes which in their individual manifestations are quite similar to those described above, but differ from them by their gradual development and by their chronic course from the start, bring with them continuously exudation of fibrin and cells and slow proliferation of young and subsequently cicatrized connective tissue, so that the latter gradually forms the principal manifestation of the macroscopic and microscopic picture of the disease. Then we have the picture of the primary productive peritonitis, pleuritis, etc. Finally, there are probably also cases of chronic pericarditis, pleuritis, etc.,

in which the exudative inflammation has never reached a higher stage, is perhaps altogether absent, and yet there has taken place a strong interstitial proliferation; as, for example, in tuberculous peritonitis, where together with tubercle formation there is a diffuse, non-exudative inflammation, perhaps conditioned by toxins.

What has been denominated hypertrophy and hyperplasia may also be ascribed to a particular method of observation. Originally the name designated the increase in volume of an organ dependent upon the addition of specific organic tissue, as is the case, for example, in compensatory or work hypertrophy or in functional hypertrophy in general: hypertrophy of the heart in increase of resistance in the circulation, hypertrophy of the muscles of the stomach in stenosis of the pylorus, the increase in volume of the muscles of the body in athletes. The cases in which the enlargement of a part was conditioned by foreign deposits or by the increase of another tissue, as, for example, of the interstitial fatty tissue in the muscles, were distinguished as pseudohypertrophy.

Here also new factors had been added to the original conception calculated to extend it and to wipe out its limits. At first medical men spoke of an hypertrophy of individual tissues, *e. g.,* one of the fatty tissue as in adiposity, of the connective tissue, as in elephantiasis, of the epithelium in wheals, cutaneous horns, etc.; finally also of hyperplasia of the interstitial connective tissue in conditions arising from acute inflammation or independently as in so-called secondary contracted kidney. As in all these cases there is an actual increase of tissue, we can speak of a pseudohypertrophy only in reference to the organ, while with regard to the hyperplasied tissue itself such a distinction is of course unwarranted. Hence, here also we have a method of observation.

If, however, we inquire into the origin of the hyperplastic formation of new tissue we are immediately confronted in many of these forms with the impossibility of distinguishing them from inflammation; a large percentage of the cases of hyperplasia, for example, of interstitial tissue, and partly also of the glands in chronic catarrhs are inflammatory processes in the ordinary sense, and many other actions of irritation leading to hyperplasia as the elephantiasis of the cutaneous and subcutaneous connective tissue caused by the *Filaria sanguinis* cannot be distinctly separated from diffuse, productive inflammatory processes. There are not

only intermediate forms as in various animal and plant species, but in general there is wanting every unifying, limiting element for a differentiation of the processes. The distinction between inflammation and hyperplasia is of no value because the two designations have been chosen from quite different stand-points. We can no more compare these two processes than in zoology we can divide warm-blooded animals and aerial animals into two different classes. Both designations are of significance only from a certain point of view and have a meaning only in so far as a definite aspect is considered; they do not by any means exhaust all the peculiarities of the objects under consideration.

We shall meet with precisely the same difficulties when we attempt to distinguish hyperplastic processes from reparatory and compensatory processes, in a word, from regulative processes. When we look at them from the last-mentioned point of view we shall find that a great percentage of hypertrophies represent directly regulative processes.

Hypertrophy leads to the consideration of tumors, which again extends, on the one hand, into the field of inflammation, and, on the other, into that of hyperplasia. Certain forms of valvis, pointed condyloma, circumscribed epithelioma are forms of manifestations which cannot at all be classed under one of these three groups or perhaps may be included with equal propriety in each of them. Under genuine tumors (a name which originally only meant swellings) have been included for some time only the proliferation swellings, those in which a new formation of tissue is really the basis of the enlargement of the organ. Further, practical elements in particular were taken to distinguish, for example, hyperplasia of the connective tissue from fibroma and inflammatory proliferation; only circumscribed hyperplasia is designated as fibroma, but not diffuse proliferation. But it is evident that between a diffuse fibromatose of the entire mammary gland or of the entire ovary and a large fibroma affecting the entire organ there is really no difference, and further that certain tissue proliferations even of a diffuse variety may assume the distinct character of the large group of malignant tumors. No one will regard a diffuse cancer of the stomach as being a hyperplasia. In one case then the circumscribed or diffuse manifestation is the distinguishing element, in other cases the non-malignancy or malignancy—two distinct points of

view which have nothing whatever in common. It may be that malignant tumors and non-malignant ones differ more greatly from each other than do the latter from hyperplasia. But here also every *principium divisionis* is wanting.

I need not point out that what I have said above is true also of the so-called infiltration swellings. It is evident that a tubercle or gumma is as much an inflammatory process (in fact, these forms very often pass into a diffuse inflammation) as is a tumor, because a new foreign mass has formed which removes or destroys the organic tissue. According as the etiological or purely morphological point of view is accentuated these new formations can be regarded as "specific inflammation" or as "tumors," and both designations are used in the next-books; I need but remind you that Virchow included both the above-named forms of new formations under tumors, and that it was only with the extension of etiological observation, especially after the discovery of the tubercle bacilli, that it became more and more common to classify tuberculous processes among inflammations.

To recapitulate, we have again in the names of "reparative processes," "hypertrophy," "tumors," and "infectional swellings," only chapter headings for processes considered from a *certain point of view,* but which can in no way be regarded as a system, not even temporarily; for many of the various processes which come under one of the above headings, if regarded from another stand-point, can as well be classed under another heading. None of the chief groups to which these names are applied is all-inclusive, but contains only characteristic processes. The point of observation is the only criterion for classifying any single process.

If now we no longer designate with the name inflammation a process of disease in its entirety, but recognize the term as describing only one of the clinical phenomena, then we have but done what has always obtained in other groups of pathological processes; we have ceased to force into the conception of inflammation every possible manifestation that does not belong to it. We do in the familiar manner what you, gentlemen, are doing all the time as practitioners when you speak of an inflamed wound, or the inflammatory character of a wound-healing process, meaning that the edges of the wound show the symptoms of inflammation.

From this stand-point alone, that of regarding inflammation as

an attribute, a definite character of a disease, are we justified in saying what anatomical and clinical practice forces us daily to say in so many cases: such and such a disease has a more or less inflammatory character. And this, I believe, is not a retrogression but a necessary return upon the path of objective research.

These are, in my opinion, the conclusions which can be drawn from the present state of our knowledge of the subject of inflammation. They have more than is usual in such presentations a transitory character, but for this reason they take more account of the fact that our knowledge now as it was in the past and always will be in the future is relative. We must not, however, for this reason refrain from precisely defining our views from time to time, according to the data of a given period. I believe that with my theory of inflammation we again approach nearer to the clinical and practical field, and this is essential, for theorist and practitioner cannot forever follow different roads when they have to deal with the same subject. In conclusion, therefore, I would only ask you to test and examine my position from your own stand-point, and see in how far my critical study of the *theory* of *inflammation* can itself bear *criticism*.

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SAVES TIME AND PATIENTS

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